oks are not absolutely dead things, but do contain a potency of life in them to be active as that soul was whose progeny they are, nay they do preserve as in a il the purest efficacy and extraction of that living intellect that bred them

—John Milton

Heart Sounds and Murmurs

Heart Sounds and Murmurs

a clinical and phonocardiographic study

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Contents

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Preface

This book consists of four principal sections and an introductory chapter on technic The first section is concerned with the history and physics of sound as it relates to auscultation and phonocardiography and also with de scriptions of various recording devices commonly used in the study of heart sounds and murmurs This section chapters 2 to 6, was written by the late Mr M B Rappaport E E, of the Sanborn Company, Waltham, Massachu setts A considerable amount of this material is based on earlier papers by Mr M B Rappaport and Dr H B Sprague The second section, chapters 7 to 17, deals with auscultation and phonocardiography in acquired heart dis ease Dr H B Sprague collaborated with me in the writing of this section The material for it was gathered during the four years 1951 to 1954 when I had the privilege of working in the cardiac department of the Massachu setts General Hospital and also at the House of the Good Samaritan, Boston The third section, chapters 17 to 29 is concerned with auscultation and phonocardiography in congenital heart disease, Dr A S Nadas collaborated with me in the writing of this section The material was gathered during the three years 1954 to 1957 when I had the privilege of working in the Sharon Cardiovascular Unit at the Children's Hospital, Boston The fourth section chapters 30 to 33, contains miscellaneous information concerned principally with arrhythmias, and the data were obtained over the past 8 years

Numerous persons have assisted in many ways during the preparation of this book, but some deserve particular mention first and foremost one of my co authors the late Mr M B Rappaport, who first taught me how to take and read phonocardiograms and who patiently answered scores of questions on the physics of sound as related to auscultation and phonocardiography next the members of the cardiac department of the Massachusetts General Hospital particularly Drs P D White, H B Sprague and E F Bland who provided me with a laboratory to work in and gave me access to their patients and their records Dr A S Nadas at Children s Hospital, Boston and Dr B F Massell at the House of the Good Samaritan Boston gave me complete access to their patients and their records and Dr A M Rudolph, also at Children's Hospital was most helpful in enabling me to obtain simultaneous pressure tracings and sound recordings when this seemed to be indicated To Judith A Ashby, who spent countless hours translating articles from various foreign languages which I could not read and to Mrs Marcia Lawson, who edited this manuscript I owe my greatest thanks, for without their help this book would never have been completed

I take full responsibility for the phonocardiography tracings, their tech nical quality the accuracy or otherwise of their labelling and interpretation and for any errors and omissions in the text

PATRICK A ONGLEY

section I

history and physics

1/ Introduction

Auscultation and phonocardiography represent two complementary methods for studying heart sounds and murmurs. Auscultation is the older method and its systematic use dates from the invention of the stethoscope by Laennec[1] in 1816. Phonocardiography was developed by Einthoven[2] in 1894, although some work had been done by Chauveau and Faivre in 1855[3] and Donders in 1859[4].

As employed today, auscultation is adequate for routine clinical examination, but in the investigation of complex arrhythmias, marked tachycardias, and cardiac cycles containing multiple sounds or murmurs, phonocardiography, together with suitable reference tracings, is essential for correct interpretation.

AUSCULTATION

History

It is certain that from early time, physicians have been aware that the beating of the heart results in audible sounds. The great wonder is that it took so long for the art of auscultation to develop. Harvey[5] recognized the first heart sound in 1628, and, prior to Laennec's[1] discovery of the stethoscope, Corvisart[6] gave such admirable descriptions of the palpable thrills associated with mitral stenosis that it is difficult to believe that he too did not hear the murmurs of these patients with the ear directly applied to the chest wall. During the latter part of the eighteenth and the early nineteenth centuries Corvisart[6] and doubtless many others noted that heart beats could be heard when the ear was applied directly to the chest, that is by *immediate* auscultation. However, it was not until 1816 that Laennec invented the first crude stethoscope which he described in 1819, and which signalled the birth of *mediate* auscultation. After this events moved quickly and during the next forty years excellent descriptions of the sounds and murmurs in healthy and diseased hearts appeared in the French and English literature and, to a lesser extent, in the German and American literature.

Laennec[1] described many murmurs and sounds occurring in stenotic lesions, arrhythmias and functional cardiac states. It is well to remember, however, that Laennec attributed the second heart sound to atrial contrac

ascending limb of the c wave is helpful in determining the end of the first heart sound, but we have not found the v wave to be of any great use It must be remembered that tracings from the jugular vein reflect events in the right side of the heart, and it may be difficult to correlate these with sounds and murmurs produced in the left side of the heart

For timing the aortic valve closure, the *arterial pulse*, with its dicrotic notch, is of the greatest help, whereas for the timing of the early systolic sounds, both *intraventricular* and *intra arterial pressure tracings* from cardiac catheters often are necessary

For the investigation of atrial sounds, much work has been done with *intra esophageal tracings*

4 The recording room should be as soundproof as possible, with absolutely no talking or moving around by persons in the room while the recording is being made

5 Outside electrical interference should be eliminated as far as possible

6 Before recording a phonocardiogram on any patient, it is essential that a full cardiac examination be performed and that the findings of all the examiners be recorded This prevents physicians from making routine requests for phonocardiograms to solve their auscultatory problems without first making a conscientious and detailed clinical examination It also allows a later study of the records and a comparison of the results of auscultation and phonocardiography, in order to determine where and under what circumstances mistakes are most frequently made

Technic

Since phonocardiography is simply a refinement of auscultation it is obvious that the method of approach will be similar The method described here is that which we follow in our clinics and which we have found to be satisfactory

Certain basic recordings are made routinely, and additional recordings are made in selected cases, as required On the recording machines available to us we have the stethoscopic and logarithmic microphones (see chapter 7) Since the stethoscopic microphone records low frequency sounds, such as the third and fourth heart sounds, as well as those in the inaudible range, we begin by taking a stethoscopic recording at the apex, using the electrocardiogram as a reference tracing Then we change to the logarithmic type of recording device, and simply apply the microphone to the routine areas of auscultation (see table 1)

If, during clinical auscultation, we have decided that recordings in other areas would be useful (such as under the left clavicle in a patient who has patent ductus arteriosus over the first dorsal spine in one who has coarctation of the aorta or on the right side of the chest in one who has dextrocardia), we explore these areas with the appropriate chest pieces and make suitable recordings

tion, a fact excusable enough, but one which must be recognized in order to interpret his descriptions adequately

PHONOCARDIOGRAPHY

Requirements

Although there is no universally accepted method for recording a phono cardiogram, and, although there are many variations in technic, there are certain basic requirements These are as follows

1 A suitable recording device must be selected which fulfills the criteria outlined in chapter 6

2 It is *important that the patient be warm, relaxed, and preferably* stripped to the waist Only under these conditions can the artefacts result ing from the contact of the microphone with the patient's clothing be avoided It is also important that the chest piece be applied with sufficient firmness to prevent friction between the recording device and the patient's skin and body hair

Muscular tremor from a shivering patient, or one who is apprehensive or dyspneic, results in distortion of both the electrocardiographic baseline and the phonocardiographic tracing, making an accurate interpretation difficult

If a patient can co operate, it is best to have him hold his breath in mod erate expiration Since this is not possible in small babies and extremely sick patients, one must learn to recognize artefacts due to respiration and to make suitable allowances for them It may be helpful to have the patient voluntarily take several deep breaths and to take the tracing in the period of apnea which follows

3 There must be simultaneous recording of suitable reference tracings The easiest and most commonly used reference tracing is the *electrocardio gram* This is satisfactory for timing the first heart sound which must occur after the Q wave of the electrocardiogram, the second sound near the end of the T wave, and the atrial sound which must occur after the P wave However, it is of no use for timing the diastolic events, as there is no con stant relationship between the mechanical and electrical events of the car diac cycle during diastole

On the other hand, we have found the *apex cardiogram* to be of little use during systole, but of great assistance during diastole, in those cases in which a good, rapid, inflow wave is recorded We have learned that the open ing of the atrioventricular valves coincides with the "O" point at the begin ning of the rapid inflow wave, and that the third heart sound occurs at the summit of this wave A split second sound records its two components on or before the negative wave preceding the "O" point of the apex cardiogram

The *jugular venous pulse* is often helpful, and, indeed, is considered by Orias and Braun Menendez[7] to be the most useful of all the reference trac ings Certainly, the *a* wave is useful in timing atrial contractions, and the

TABLE 1

TYPICAL PHONOCARDIOGRAPHIC RECORD CARD

Patient's status

Name	*Age*	*Sex*	*Occupation*	*Clinical diagnosis*

Reason phonocardiogram requested

Position	Supine	Supine	Supine	Supine	Supine	Supine	Supine	Sitting forward and to left
Respiration (arrested expiration)								
Area	Apex		4 LIS	2 LIS	2 RIS	Apex	4 LIS	3 LIS
Microphone	Steth	Log	Log	Log	Log	Log	Log	Log [illegible]
Chest piece	MOB	MOB	MOB	MOB	MOB	MOB	MOB	Black diaphragm
Pulse	Apex cardiogram	Apex cardiogram	JVP	JVP	JVP	JVP	JVP	JVP
ECG Lead	II	II	II	II	II	II	II	II
Amplitude	6	6	6	6	6	6	6	6

M O.B signifies the medium open bell
J V.P signifies the jugular venous pulse
The black diaphragm is 0 015 in thick.
The "brown or thick diaphragm is 0 035 in thick

additional record in position 7 (with the patient sitting), using a diaphragm chest piece to record more clearly a diastolic murmur at the left sternal border

If a two-channel machine is being used, the same system is followed, except that it is not possible to record both a pulse tracing and an electrocardiogram simultaneously with the phonocardiogram, therefore, extra tracings must be made This means that more film is used, but the two channel machine is easier to operate, and, especially for children, the two beams are easier to control than are the three beams of the tri beam machine

Analysis and Interpretation of the Phonocardiogram

The phonocardiogram (Fig 1) is interpreted as follows

1 First the individual heart sounds are described noting their intensity

The routine method of recording in each area is as follows

1 Explain the procedure to the patient in some detail to allay apprehension and to gain the patient's co operation

2 Make sure the patient is warm, relaxed, and comfortable

3 Apply the electrocardiographic electrodes or the appropriate pulse attachments

4 Choose the stethoscopic or logarithmic recording device, as required, and choose the appropriate chest piece, such as the small, medium, or large open bell, or the thin or thick diaphragm (see figure 10)

5 If possible, apply the microphone to the chest wall with a rubber strap encircling the chest, being careful to adjust the tension of the strap in order to obtain the best identification of the heart sounds For children and extremely sick patients, it is easier to hold the microphone in position by hand

6 Having allowed the machine to warm up, and while listening with the amplifying stethoscope, make certain there are no extraneous sounds, and examine the recording beams on the machine to determine whether they are adequately spaced and will give a stable baseline In addition, make certain that the sound vibrations are of sufficient amplitude to give a good tracing which will be easy to interpret

It is a mistake to insist on a perfectly flat baseline, since, in order to achieve this, some murmurs of low intensity and either high or low frequency may be screened out One soon becomes adept at differentiating any artefacts, or fuzziness of the baseline, from the true murmurs

After having explored the routine areas of auscultation with the electrocardiogram as a reference tracing, we routinely record the apex logarithmic, and often the apex stethoscopic, phonocardiogram with the linear apex cardiogram Depending on which additional sounds or murmurs interest us particularly, we next record the heart sounds in the corresponding areas, with the jugular venous pulse tracing being recorded simultaneously

In choosing the recording chest pieces, we routinely use the open bell, with the size appropriate to the patient's chest, and then use the diaphragm for any areas where we consider it necessary

This completes the routine phonocardiogram, but, depending on the patient and the murmurs and sounds which we are recording, we adopt additional methods comparable to those which are used in auscultation To accentuate apical rumbles, we exercise the patient, turn him in the left lateral decubitus position, and apply the bell lightly to the apex To accentuate aortic diastolic murmurs, we often have the patient sit up, leaning forward and to the left, while a diaphragm chest piece is applied firmly over the aortic area or in the third or fourth left interspace

As the phonocardiogram is taken, a record is made of all the procedures performed (Table 1)

This chart shows a patient's typical phonocardiographic record in positions 1 to 6, the areas usually recorded in a routine phonocardiogram, with an

TABLE 1

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Patient's status								
Name			*Age*	*Sex*	*Occupation*		*Clinical diagnosis*	
Reason phonocardiogram requested								
Position	Supine	Supine	Supine	Supine	Supine	Supine	Supine	Sitting forward and to left
Respiration (arrested expiration)								
Area	Apex		4 LIS	2 LIS	2 RIS	Apex	4 LIS	3 LIS
Microphone	Steth	Log	Log	Log	Log	Log	Log	Log
Chest piece	M O B	M O B	M O B	M O B	M O B	M O B	M O B	Black diaphragm
Pulse	Apex cardiogram	Apex cardiogram	J V P	J V P	J V P	J V P	J V P	J V P
ECG Lead	II	II	II	II	II	II	II	II
Amplitude	6	6	6	6	6	6	6	6

M O B signifies the medium open bell.
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ANALYSIS AND INTERPRETATION OF THE PHONOCARDIOGRAM

The phonocardiogram (Fig 1) is interpreted as follows

1 First, the individual heart sounds are described, noting their intensity

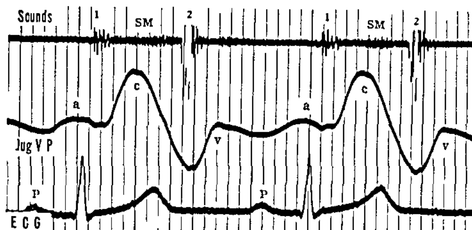


Fig 1 (2 R I S Log) Phonocardiogram of normal patient showing the a c and v waves of the normal jugular venous pulse Note the normal first sound (1) 0 06 sec after the onset of the Q wave the low intensity medium frequency rather late systolic murmur (SM) and the second sound (2) split by 0 02 sec with the first or aortic component more prominent than the second or pulmonic component The a c and v waves of the jugular venous pulse are clearly shown

at various areas, any delay of the first heart sound relative to the QRS complex, the degree, if any, of splitting of the first and second heart sounds, variations of heart sounds due to arrhythmias or other causes, the presence or absence of the third or fourth heart sounds, any additional sounds, and any other relevant features

2 Next, the systolic murmurs are described, noting their relative frequency and intensity, and also the shape of the murmur (plateau, diamond, descrescendo)

3 Then the diastolic murmurs, if any, are described, noting their relationship to the second, third, and first heart sounds Any unusual features in the pulse wave or the apex cardiogram are also noted and described, and finally a brief summary of the relevant features is made

Thus, there are two separate records of the patient's heart sounds and murmurs, the auscultation record and the phonocardiographic interpretation Often a comparison of these helps to explain arrhythmias, rapid tachycardias, or exact timing of murmurs and gallops (see chapters 8 and 30)

It is important to stress that a clinical diagnosis should not be made from a phonocardiogram alone It is true that, just as one tends to recognize electrocardiographic patterns as being associated with certain clinical states certain phonocardiographic patterns are typical of specific clinical states However, it must be emphasized that a phonocardiogram is purely an accessory method in cardiac examination, and only with this understanding can the tracing be interpreted accurately

ARTEFACTS In the interpretation of phonocardiograms, particularly those taken early in one's experience or on patients who either will not or cannot co operate, artefacts of various types will be encountered In this respect

the phonocardiograph is inferior to human auscultation The human cortex is selective and, with experience, learns to concentrate on certain sounds or murmurs, or on certain phases of the cardiac cycle, excluding all extraneous sounds The recording device on the other hand, records all the sounds within its range of frequency, irrespective of their origin

Most common among the artefacts encountered are the vibrations due to respiration In a small baby who is breathing rapidly, and especially if it is crying as well, it is almost impossible to take a readable phonocardiogram In dyspneic or tachypneic adults, the tracing may be difficult to interpret, but, if sufficiently long strips of tracing are taken, there are usually enough cardiac cycles free from artefacts to make an interpretation possible Breath sounds can be identified easily on the phonocardiogram by their asynchronism with the cardiac rhythm and by one's knowledge of the patient's condition during the recording

Muscular tremor in excited, tense or thyrotoxic patients or in those with certain neurologic disorders such as Parkinson s disease, results in considerable irregularity of the baseline throughout systole and diastole Usually this irregularity is reflected in the electrocardiogram as well, and therefore, can be easily identified

Certain additional sounds such as borborygmi or the rubbing of the recording microphone on the hair of the chest may introduce additional vibrations These can be recognized by the fact that they are not repeated from one cycle to another Since one listens to the heart sounds while the phonocardiogram is being recorded one should as an additional precaution make a note of any adventitious sounds

IMPORTANT CONTRIBUTIONS TO CLINICAL AUSCULTATION

1628 Harvey[5] described the first heart sound which he thought was due to the shock of the cardiac apex against the chest wall during systole

1819 Laennec[7] having discovered the stethoscope made a series of brilliant observations on murmurs associated with stenotic lesions regurgitant lesions and so-called functional murmurs

1822 Lejumeau de Kergaradec[8] described fetal sounds and placental murmurs

1824 Collin[9] described the friction rub associated with pericarditis

1829 Williams[10] described atrial sounds in a case of bradycardia

183 Hope[11] described the murmur and thrill associated with dilatation of the pulmonary artery and pulmonic stenosis

1832 Corrigan[12] described the Corrigan pulse in free aortic regurgitation

183 Rouanet[13] attributed the second sound to the closure of the semilunar valves

1835 Williams[14] differentiated between the diastolic murmurs of mitral stenosis and aortic regurgitation

1835 Bouillaud[15] described triple and quadruple rhythms

1838 Charcellay[16] described a presystolic sound in Bright s disease

1843 Fauvel[17] described the presystolic murmur of mitral stenosis

1862 Flint[18] described a presystolic murmur associated with aortic regurgitation

186 Duroziez[19] described Ffout ta ta Rou in mitral stenosis

1866 Potain[20] described normal splitting of the first and second heart sounds

1875 Potain[21] described the gallop rhythms

1879 Roger[22] described the systolic murmur associated with small ventricular septal defects

1888 Steell[23] described the pulmonic regurgitant murmur associated with hypertension of the pulmonary artery

1888 Rouches[24] described the opening snap of the mitral valve in mitral stenosis

1900 Gibson[25] described the continuous murmur of patency of the ductus arteriosus

1905 Obrastzow[26] and (in 1907) Gibson[27] and Hirschfelder,[28] all independently described the third heart sound

1906 Straschesko[29] described the 'bruit de canon' associated with complete heart block

1923 Castex[30] described the splitting of the first and second heart sounds in bundle branch block

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2 / History of the Stethoscope

PRESTETHOSCOPE ERA

Auscultation, a method of investigation of the functions and conditions of the respiratory, circulatory, digestive, and other organs by the sounds they themselves produce, or that are elicited by percussion, is one of the most ancient modes of diagnosis From the classic passage in Hippocrates' work, "de Morbis," "If you listen by applying the ear to the chest," we have definite evidence that immediate auscultation was employed more than twenty centuries ago

Harvey's dissertation, "de Motu Cordis," gives convincing information that the sounds of the heart had not escaped him

> It is easy to see when a horse drinks that water is drawn in and passed through the stomach with each gulp the movement making a sound and the pulsation may be heard and felt So it is with each movement of the heart when a portion of the blood is transferred from the veins to the arteries that a pulse is made which may be heard in the chest

Corvisart [1] made the following observation

> Some authors assert that they could hear in certain diseases of the heart the noise produced by the violent strokes of this organ even at a small distance from the patient s bed I have never had an opportunity I repeat it of ascertaining the unquestionably rare observations I have barely heard these strokes by applying my ear close to the patient s thorax

However, during the prestethoscope era physicians derived little benefit from auscultation other than that of sensing the pulsations of the heart by the mere application of the hand

THE LAENNEC STETHOSCOPE

"I was consulted," says Laennec,

> in 1816 by a young woman who presented some general symptoms of disease of the heart in whose case the application of the hand and percussion gave but slight indications on account of her corpulency On account of the age and sex of the patient the common modes of exploration being inapplicable I was led to recollect a well known acoustic phenomenon namely if the ear be applied to one extremity of a beam a person can very distinctly hear the scratching of a pin at the other end I imagined this property of bodies might be made use of in the present case I took a quire of paper which I rolled together as closely as possible and applied one end to

the precordial region by placing my ear at the other end I was agreeably surprised at hearing the pulsation of the heart much more clearly and distinctly than I had ever been able to do by the immediate application of the ear

Laennec experimented further with his tube and finally evolved the instrument known as the "stethoscope"

> This consists simply of a cylinder of wood a foot in length perforated in its center longitudinally by a bore three lines * in diameter and formed so as to come apart in the middle for the benefit of being more easily carried *One extremity of the cylinder* is hollowed out into the form of a funnel to the depth of an inch and a half which cavity can be obliterated at pleasure by a piece of wood so constructed as to fit it exactly with the exception of the central bore which is continued throughout it so as to render the instrument in all cases a pervious tube *The complete instrument* that is with the funnel shaped plug infixed is used in exploring the signs obtained through the medium of the voice and the action of the heart the other modification or with the stopper removed is for examining the sounds communicated by respiration A solid cylinder without any perforation is the best *instrument for exploring the* action of the heart but as this form is not so good for examining the voice and respiration the perforated cylinder is commonly used for all purposes

Having developed a suitable stethoscope, Laennec undertook his epoch making observations

> I commenced immediately at the hospital Necker a course of observations which have resulted in the discovery of new signs ure for the most part obvious easy to be possessed of and suitable to render the diagnosis of almost all diseases of the lungs the pleuras and the heart more certain and perhaps more circumstantial than even the surgical diagnostic signs established by the aid of the probe or the finger[3]

Piorry[4] in 1828 modified the Laennec stethoscope by reducing it to the thickness of a finger He constructed an ear piece which overcame the difficulties experienced by some physicians in obtaining a proper seal to the ear Piorry s modification included a trumpet shaped chest piece Essentially, his instrument is the modern monaural stethoscope

C J B Williams[5] a student of Laennec's further modified Piorry's stethoscope by reducing the sharp rim of the chest piece and making the ear piece movable to facilitate auscultation above the clavicle and scapula

Many years later it was observed that it was not necessary to have a stethoscope with a rigid wall such as the Laennec, Piorry, or the Williams types and that the sounds could be heard equally well with an India rubber tube As a result the monaural flexible stethoscope came into existence

THE BINAURAL STETHOSCOPE

The exact date of the first binaural stethoscope is not known, several people have been credited with this invention In 1907, C T Williams,[6] in

* A line is equal to 1/12 part of an inch

a lecture delivered before the Medical Department of the University of Oxford, brought to light some rather interesting historical information pertaining to the binaural stethoscope He stated, "The late Dr Leared claimed to have invented the double stethoscope in 1851 I present for your inspection a curious specimen of a binaural stethoscope which my father constructed in 1829, and which is probably the first binaural stethoscope ever made" The C J B Williams binaural stethoscope consisted of a trumpet shaped chest piece of mahogany, the end of which was screwed into a connection to which were attached two bent lead tubes which could be adjusted to the ears The lead tubes had no ear pieces or fittings of any kind and were difficult to apply At the time the C J B Williams binaural stethoscope was devised, India rubber tubing had not been introduced

At the International Exhibition of 1851, Leared[7] showed a binaural stethoscope which he claimed to be an improvement over the monaural stethoscope It consisted of two guttapercha tubes attached to the chest piece at one extremity and to the ear pieces at the other These tubes, being drawn apart and applied to the ears, were kept in place by their own elasticity

In 1852 Cammann[8] modified the Leared binaural stethoscope, by improving the ear pieces and by making it lighter in weight and more convenient for carrying Snelling[9] attached a rim of soft rubber to the chest piece and claimed that it was easier to apply since it conformed to the unevenness of the chest

It was not until the invention of the diaphragm chest piece with a rigid covering over the end of the collecting part that a significant modification of the older bell type was developed Marsh,[10] in 1851, patented a stethoscope with a flexible membrane stretched over the end, but the phonendoscope devised by Bianchi,[11] in 1894, was the first instrument to have a rigid diaphragm The chest piece of this type, which is in common use in this country at the present time, is the one patented by R C M Bowles[1] in 1901 The diaphragm chest piece attenuates the low pitched heart sounds relative to the higher pitched murmurs such as are characteristically heard in the presence of slight aortic regurgitation, allowing the murmur to be heard more easily

In 1926, one of us (Sprague)[13] described a combination diaphragm and bell chest piece with an adjustable valve for selecting the desired chest piece About the same time Rieger[14] patented a similar stethoscope Robertson[15] and Landouzy[16] devised multiple stethoscopes by connecting a number of binaural tubes to one sound collector These instruments were useful for teaching and group listening

It would seem to be agreed at the present time that the bell chest piece and the diaphragm chest piece can satisfactorily cover the acoustic ranges necessary for clinical examination of the heart However, the development of modern electronic amplifying systems with their freedom from back

ground noise, has opened a new field for the graphic registration of heart sounds and murmurs, as well as respiratory sounds, which may well modify our clinical opinion in the future

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3 / Characteristics of the Human Ear in Auscultation

In auscultation, the human ear may be considered as the recording mechanism of an acoustic system Arnold,[1] in his introduction to Fletcher's "Speech and Hearing,"[2] which is considered one of the best treatises on the subject from a physical standpoint, states

> Our ears are only machines to translate air waves into a form suited to stimulate the auditory nerve, and as machines we may measure and describe them in the same terms that apply to devices we ourselves construct We may compare them as to performance and may accommodate our devices *to their requirements But to under* stand the mechanism of the ear is by no means to understand the act of hearing for we have not heard until the brain has perceived the message sent by the auditory nerve We cannot explain in precise mechanical terms how this is done nor indeed have we any clear comprehension of the process at present Some important factors relating to the process of hearing we can however determine by measuring the least changes in sound which can be detected under a variety of conditions of pitch loud ness and accompanying noise Thus we may obtain a quantitative means of compar ing individuals in this respect and establish a standard of average hearing

THE ORIGIN AND PROPAGATION OF SOUND

When a vibratory motion is allowed to communicate with the ears, audi tory sensations may be produced Auditory stimulation is most commonly produced by sound waves in air, although other mediums such as water can produce similar effects If, for example, an object is set into a to and fro or simple harmonic motion in an elastic medium such as air, the physical movement of the object produces a compression in adjacent air on one side and a rarefaction on the other The molecules of air in the compressed region are immediately propelled toward neighboring molecules, which in turn transmit pressure impulses to more remote molecules, and so on In this way a pressure impulse moves away from the vibrating object at a definite speed When the vibrating object retreats, it draws the nearby molecules of air toward it, producing a partial vacuum which is immediately filled by molecules further removed In this manner a rarefaction impulse is pro duced, which travels in the opposite direction and at the same speed as the pressure impulse

A given molecule of air in the path of the alternating pressure and rare faction impulses or waves undergoes a to and fro or simple harmonic motion which simulates the vibratory motion of the object but shows a temporal

lag which grows progressively greater with distance Although the pressure impulse propagates away from the vibrating object at a definite speed, the molecules of air do not travel with the pressure impulse Furthermore, since the alternating pressure impulses spread as they travel away from the source of excitation they weaken as the distance is increased

A phenomenon which is often used as an analogy to the action of sound waves occurs when a stone is dropped into a smooth pond Water is carried down by the stone and a disturbance is propagated through the water in the form of a wave which travels in all directions at a definite velocity However, the molecules of water do not travel with the waves, as may be indicated by corks bobbing in a pool There is a significant difference between this type of wave motion and that of sound The molecules of water vibrate up and down in a direction which is transverse to the direction of wave travel, rather than back and forth along the wave path as occurs when an object vibrates in air The surface water wave is classified as a *transverse* wave and a sound wave as a *longitudinal* wave

GRAPHIC REPRESENTATION OF WAVES

The characteristic properties of a transverse water surface wave may be graphically *represented* by drawing a cross sectional view The graphic representation of a longitudinal wave is more complex as the magnitudes of the alternating compressed and rarefied molecules of the stimulated medium cannot be adequately *shown* in cross sectional views In sound work, we must abandon pictorial representation and substitute a graph such as figure 2 This graph represents the *pressure* variations of the air over a period of time at a given point which is at a distance from the vibrating object

The axis of ordinates of figure 2, represents the air pressure, *which is*

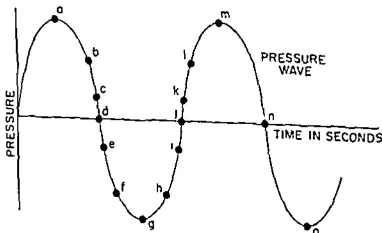


Fig 2 A graph which is symbolic of a sound wave in air

usually expressed in dynes * per square centimeter or microbars, and the axis of abscissas indicates time in seconds The maximum amplitude of the wave as it deviates from the horizontal axis is a measurement of the amount the air is compressed or rarefied at the fixed reference point An upward deviation or peak of the pressure wave represents compression and a negative dip or trough represents rarefaction, where the wave crosses the horizontal axis, the medium is in an undisturbed or normal state The greater the magnitude of compression and rarefaction of the air, the greater is the pressure that would be applied to the tympanum of the ear and the greater would be the perceived loudness

The graph also shows that in this case the air at the reference location undergoes pressure variations in a simple harmonic pattern, or *sinusoidally*, and that points a to i represent different phases of compression or rarefaction Points a and m differ by 2π radians † or 360 degrees, points a and d by $\frac{\pi}{2}$ radians or 90 degrees, and points a and g by π radians or 180 degrees Points a and m are in the same phase of excitation of the pressure wave and the distance between them is a wave length

WAVE FREQUENCY

The frequency of the wave is the number of complete waves or cycles, such as is measured in figure 2 from a to m, d to n, or g to o, which pass the reference location in one second Thus, the frequency of a wave is generally expressed in *cycles per second* The auditory sensation of pitch depends on the frequency of the wave, the higher the frequency, the higher is the pitch All sounds do not possess pitch, as is evident from listening to everyday sounds of the street Pitch requires that a number of successive cycles of the same frequency be repeated Regularity or repetition of successive cycles makes the wave a *periodic* one Periodic waves are generated by such devices as tuning forks and musical instruments, whereas nonperiodic waves, or noises, are produced by the rustle of leaves in the wind the jangle of keys, or the noise of a typewriter

WAVE VELOCITY

The velocity of a wave is defined as the rate with which a particular phase of pressure moves along If n pressure waves pass a point in one second, and each has a wave length of λ the velocity or distance traversed by the wave in one second is

$$v = n\lambda \qquad (1)$$

* A dyne is a measurement of pressure 980 dynes are equal to one gram One dyne per square centimeter is equal to one microbar

† A radian is an arc whose length is equal to the radius of the circle of which it is a part

The velocity at which a compressional wave propagates depends on the elasticity and density of the medium Newton in 1686 suggested that the velocity, v, of a wave of compression or expansion in a medium of which the volume elasticity is E and the density d is expressed by the formula

$$v = \sqrt{\frac{E}{d}} \tag{2}$$

According to Newton s formula, the velocity of a compressional wave depends directly on the square root of the elasticity of the medium and inversely on the square root of its density Therefore, the velocity of a compressional wave is greater in a medium of low density and high elasticity In gases at constant temperature the elasticity is equal to the gas pressure if the pressure change is extremely small However, Newton observed that if pressure was substituted for elasticity in formula 2, the calculated velocity was about twenty per cent lower than the measured value Newton sought to account for this discrepancy by a number of ingenious suppositions which were physically unjustified but his formula is valid for liquids and solids

It was not until 1816, one hundred and thirty years later, that Laplace[3] observed the error in Newton s formula and corrected it Laplace suggested that although the average temperature of air is unchanged by the passage of sound waves, yet, in the compressed portion of a wave the air is instantaneously heated and during rarefaction the air is instantaneously cooled These temperature changes take place so rapidly that there is not sufficient time for heat flow to take place and for all practical purposes, an adiabatic condition exists Instantaneous heating during compression resists compression and instantaneous cooling resists expansion, and as a result, the effective elasticity is increased by a factor of γ, which is the specific heat of the gas at constant pressure to that at constant volume To determine the velocity of sound in diatomic gases such as air in which γ is 1 4, formula 2 becomes

$$v = \sqrt{\frac{1.4\,p}{d}} \tag{3}$$

where p is the pressure The velocity of sound in common mediums is given in table 2

WAVE FORM

The wave form of figure 2 is the simplest form of vibration produced by simple harmonic motion, which physicists and mathematicians call a *sine wave* A vibration of simple harmonic motion, such as is produced by a tuning fork, creates pressure waves in air of a sinusoidal configuration or wave form which is perceived by an observer as an unmusical sound Musical sounds of various instruments are associated with characteristic

TABLE 2

THE VELOCITY OF SOUND IN COMMON MEDIUMS

Medium	*Meters Per Second*	*Feet Per Second*
Air at 0 C	331	1 087
Hydrogen at 0° C	1,286	4 219
Carbon Dioxide at 0° C	261	856
Water at 13° C	1 441	4 728
Brass rod	3,600	11 800
Iron rod	4,950	16 240
Steel rod	5 000	16 410
Pine wood along the grain	3,300	10 830

wave forms which can be differentiated readily by oscillographic means or hearing

Figure 3A is a sinusoidal pressure wave in air, which was produced by the vibration of a tuning fork Figure 3B is the characteristic wave form of a violin producing a note of exactly the same frequency and amplitude as the tuning fork, which is heard as a sound with a musical quality and is easily identified by both oscillographic means and hearing Figure 3C is the charac

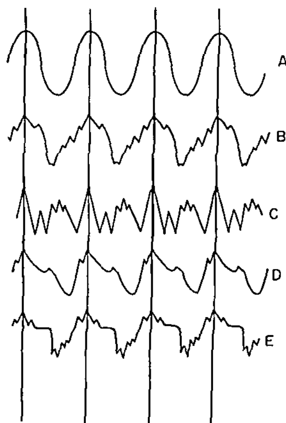


Fig 3 Wave forms of equal frequency but different configuration as obtained from A a tuning fork B the violin C an oboe D the bass flute and E the clarinet

teristic wave form of an oboe figure 3D is a bass flute, and figure 3E of a clarinet at the same frequency and amplitude as the tuning fork and violin The oboe bass flute, and clarinet produce distinct musical sensations to the ear as does the violin, and one instrument can be differentiated from another by hearing or wave form observation

Rather complex wave forms such as are produced by several musical instruments playing together or even those of a symphonic orchestra may be represented graphically The wave form of the most complex combination of sounds is a single line graph such as those of figure 3 Thus, the single line oscillogram of the symphonic orchestra, besides giving exact information a to the instantaneous pressure state of the air, may also be considered as representing the instantaneous di placement of the tympanums of the ears because of the applied air pressure No matter how many instruments are generating sound pressure in the air, the tympanums of the ears can be in only one position at any particular instant Since we are capable of distinguishing many instrumental sounds at one time, the complex single line wave form must represent the combination of sounds produced by the orchestra

From figure 3 it is apparent that musical instruments vibrate in complex ways in addition to vibrating at the frequency which corresponds to the musical note on the score the instrument vibrates at many higher frequencies The basic frequency as indicated by the score is called the *fundamental frequency* and the higher frequencies are called the *overtones* It is the particular combination of the fundamental and higher frequencies, in number kind and relative amplitude, that determines and distinguishes the wave form of that particular instrument

In most musical instruments the frequencies of the overtones are multiples of the fundamental frequency these overtones are commonly known as *harmonics* If the fundamental tone or frequency is A above middle C which is 440 cycles per second, the second harmonic is 880 cycles per second the third harmonic is 1 320 cycles per second, the fourth harmonic is 1,760 cycles per second and so forth

Not all musical instruments have overtones that are harmonic Strike tones such as are produced by the bass drum cymbals triangle and other percussion instruments produce overtones that are not harmonic and the unrelated frequencies do not produce a sensation of one pitch Other orchestral instruments have weak and even inaudible fundamental frequencies The lowest strings of the piano fall into this category Nevertheless, we can identify all of these instruments by their frequency structure in an oscillogram or by hearing A rather interesting phenomenon is the well known fact that when tones from instruments such as the double bass or organ are reproduced by small radios or phonographs which cannot respond to the low frequency fundamentals, the instruments can still be recognized from their harmonic content

WAVE FORM ANALYSIS

Von Helmholz[4] showed that a complex sound can be analyzed He con structed a set of hollow spheres, each of which resonated to a different fre quency When a complex sound was allowed to communicate with the resonators, certain of the resonators were activated By noting which resona tors responded and to what extent, Von Helmholz was able to show that a complex wave form consists of a summation of sinusoidal waves of different frequencies and magnitudes By setting up a series of tuning forks, each producing a sinusoidal wave such as was present in the complex wave, the complex wave form could be synthesized

There are instruments now available, known as harmonic analyzers, which can evaluate the complete structure of the most complex wave form The general principle of operation of a harmonic analyzer is to convert the sound wave into an equivalent electrical wave by microphone and amplifier tech nics The equivalent electrical wave form is then passed through tuned or electrically resonant electrical circuits The electrical components which pass through each tuned circuit and the voltage magnitude, as measured with a voltmeter, give the desired parameters of the sinusoidal components of the complex wave form *Fourier's theorem* in mathematical physics ex presses a complex wave form by stating that any periodic wave with a fundamental frequency, n, can be reduced to a set of simple harmonic fre quencies whose frequencies are n, 2n, 3n, 4n, 5n, and so forth Actual mathe matical expressions can be formulated to represent the most complex wave forms on the basis of Fourier's theorem

THE MUSICAL PITCH INTERVAL

The keyboard of a piano is divided evenly as far as pitch is concerned The same apparent increase in pitch is produced by going from middle C to the next higher C, then to the following C, and so on, or from C to D to E The musical interval from C to C is called an *octave*, whereas the interval from C to D, or D to E, is called a *whole tone* The interval from C to C sharp is known as a *half tone*

These apparently even divisions on the piano keyboard do not correspond to even divisions in frequency As the musical pitch is increased octave by octave, equal increments of cycles per second are not added but, instead the frequency is doubled If we take 440 cycles per second as a reference one octave up is 880 cycles per second two octaves up is not 880 plus 440 or 1,320 cycles per second, but 880 multiplied by 2 or 1,760 cycles per second A musical interval thus represents a geometric ratio of frequency and not a given number of cycles An octave at the lower end of the piano keyboard may cover a frequency spread of only 27 5 cycles per second, while an octave at the upper end of the keyboard may have a frequency

spread of 2,093 cycles per second The range of musical pitch for an interval of one octave at both ends of the piano keyboard is the same in spite of the marked difference in the frequency spread because this is the way the human auditory mechanism perceives sound

THE DECIBEL

The human auditory mechanism is capable of functioning over an enormous range of sound pressure or sound energy variations To express this property of hearing in quantitative terms of pressure or energy would require the use of numbers of such magnitude that their arithmetical manipulation and visualization might be rather difficult The communications engineer was confronted with this problem and overcame it by employing the decibel system of measurement which is based on a logarithmic characteristic of human hearing and is handled similarly to the musical pitch interval on the piano keyboard The decibel is a unit of ratio, as is the musical pitch interval, except that the decibel refers to sound pressure or sound power and the basic manipulation factor in the decibel system is 10 rather than 2

The bel which is named after the inventor of the telephone is essentially a logarithmic unit, or the common logarithm of the ratio of two values of energy, but can also be applied to two sound pressures, two currents, or two voltages, which are related to energy by a square law The decibel, as the term indicates is one tenth of the bel The number of decibels in the ratio of two sound pressures is 20 times the logarithm of the ratio If we let N equal the number of decibels, E_1 and E_2 the energies and P_1 and P_2 the pressures then

$$N = 10 \log \frac{E_1}{E_2} = 20 \log \frac{P_1}{P_2} \tag{4}$$

In the decibel scale relative sound intensities are always expressed in terms of differences in decibels never as a ratio of decibels or a percentage change in decibels From the definition of the decibel one decibel may represent a sound pressure ratio equal to the twentieth root of 10, or a ratio of 1 12 to 1 That is, two sounds which differ by one decibel have their pressures in the ratio of 1 12 to 1 Similarly two sounds which differ by 20 decibels have their pressures in the ratio of the twentieth power of 1 12, or 10 to 1 If a sound pressure level is doubled, the change in decibels is twenty times the logarithm of 2, which is 6 decibels Thus, doubling a given sound pressure always increases by 6 decibels the original level, no matter what the value of the original level was

Characteristic of human hearing is that equal variations of sound intensity along a logarithmic or decibel scale approximate similarly equal variations of loudness as they are perceived by a normal human auditory mechanism

Also, a one decibel variation in intensity of sound is approximately the minimum change that may be detected by the average human being

In order to obtain a better conception of the relative values in a decibel scale, let us consider table 3 In the application of the decibel scale to the measurement of the intensity of sound, a reference point, such as the thresh old of hearing, is commonly employed Table 3 lists the average decibel

TABLE 3

AVERAGE DECIBEL EQUIVALENTS ABOVE THE HEARING THRESHOLD OF VARIOUS SOURCES OF NOISE

(Survey by New York City Noise Abatement Commission)

Source of Description of Noise	*Distance from Source (Feet)*	*Noise Level (Decibels)*	*Noise Level (Amplitude Ratios)*
Threshold of pain		130	3 162 000
Hammer blows on steel plate (almost painful)	2	114	501 200
Riveter	35	97	70 790
Elevated train	15 20	90	31 620
Average motor truck	15 20	75	5 620
Busy street traffic	15 75	68	2 512
Average automobile	15 50	66	1 995
Ordinary conversation	3	65	1 778
Rather quiet street (residential)	15 300	58	794 0
Quiet automobile	15 50	50	316 2
Average office		47	223 9
Noisy residence		45	177 8
Very quiet radio in home		40	100 0
Average residence		32	39 8
Average whisper	4	20	10 0
Rustle of leaves in gentle breeze		10	3 1
Threshold of hearing		0	1 0

equivalents above the hearing threshold of various sources of noise The instantaneous values may vary plus or minus 10 decibels from the values given The first three columns were obtained in a survey by the New York City Noise Abatement Commission The fourth column was added by us to illustrate the enormous range in intensity, in more familiar terms, that exists between the threshold of hearing and the threshold of pain Column four is merely an expression of column three, converted into equivalent intensity ratios, with the threshold of hearing as the reference value

EARDRUM OR TYMPANUM MOVEMENT AT THRESHOLD

In 1935, Wilska succeeded in measuring the magnitude of movement of the eardrum at the threshold of audibility for various frequencies of

stimulation The problem of measurement was difficult because of the extremely small mechanical *movements involved* Wilska used an oscillator driven electrodynamic mechanism similar to that used in loud speakers, which was rigidly attached to the head of the *subject A tiny probe* was allowed to communicate between the moving coil of the electrodynamic transducer and the eardrum By measuring with a microscope the amount the probe moved for a known amount of current in the transducer coil, Wilska was able to express the magnitude of probe movement at various frequencies It was further established that the probe moved in direct proportion to the magnitude of current from the oscillator down to displacements equivalent to less than one thousandth of a wave length of light Thus, by measuring the flow of current in the coil, the corresponding movement *of the eardrum* was evaluated at the *threshold of hearing* for different frequencies The results of Wilska s measurements are shown in figure 4 At

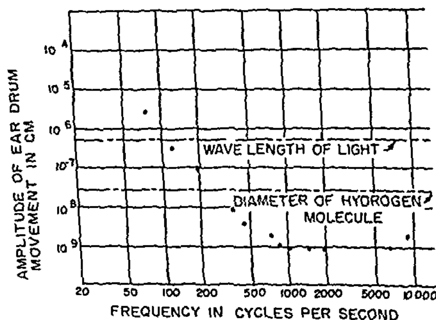


Fig 4 The amplitude of vibration of the ear drum at the threshold of hearing as measured by Wilska[6]

approximately *3 000* cycles per second, it takes a minimal amount of ear drum displacement (somewhat less than 10^{-9} cm or about 0 01 times the diameter of an atom of hydrogen) to produce a minimal perceptible sound

THRESHOLD OF HEARING AND FEELING VERSUS FREQUENCY

Pure tones of different periods of oscillation or *frequency*, but of similar *intensity* affect the human auditory system to different degrees Investiga

tions which ascertained the actual intensity of sound at the threshold of audibility were made by Fletcher,[2] Toepler and Boltzman,[6] Rayleigh, Wead,[8] Wein,[9] Abraham,[10] and Kranz [11] Wegel [12] investigated the intensity of sound at the threshold of feeling

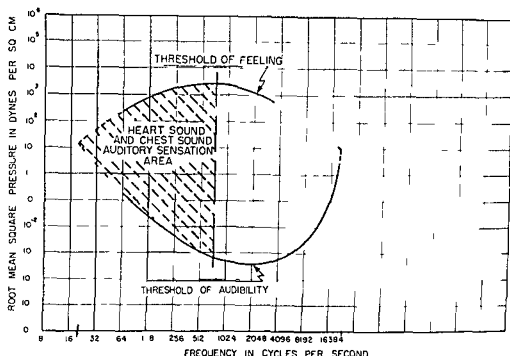


Fig 5 Average normal threshold curves of audibility and feeling (Modified from Fletcher by Rappaport and Sprague)

The curves in figure 5 represent average thresholds of feeling and audibility obtained by Fletcher - The threshold of audibility curve represents the effective or root mean square * pressure variations in dynes per square centimeter which are required to produce a sound of minimum audibility between the frequency limits of approximately 20 and 20,000 cycles per second The threshold of feeling curve represents the pressure variations beyond which a sensation of pain is created The area enclosed by the upper and lower curves represents the auditory sensation area The threshold curves of any one person would not be so smooth (peaks and valleys are superimposed), for two sets of ears are seldom identical The broken portions of the feeling and audibility curves are obtained by extrapolation, which is necessary because the phenomena are not clearly defined in these regions The shaded area represents the cardiovascular auscultation region with which we are concerned

* The effective or root mean square pressure of a sinusoidal pressure wave is equal to 0 707 times the maximum or peak pressure attained by the wave If the wave is made up of a fundamental and harmonics the effective value of the complex wave is the square root of the sum of the squares of the effective values of the fundamental wave and all the harmonic waves In alternating voltage measurements most voltmeters unless they are called peak voltmeters measure the root mean square voltage and not the peak voltage

By relating figures 4 and 5, it is possible to evaluate the relationship between the magnitude of eardrum vibration and the number of dynes per square centimeter of sound pressure needed to produce threshold auditory sensations The extremely small amount of acoustic pressure necessary to produce the threshold sensation of sound brings up an interesting question as to whether the limiting factor in the hearing of minimal level sounds lies in the anatomy and physiology of hearing [13, 14] or in the physical properties of air as a transmitting medium

It is known that the molecules of air are in constant random movement or agitation which is related to temperature This phenomenon, known as the Brownian movement, produces a spectrum of thermal acoustic noise comprising multitudinous unrelated frequencies In 1933, Sivian and White [15] experimentally evaluated the pressure magnitudes of these thermal sounds between 1 000 and 6 000 cycles per second They observed that throughout the measured spectrum the root mean square thermal noise pressure was about 86 decibels below one dyne per square centimeter The minimum root mean square pressure that can produce audible sensation between 1,000 and 6 000 cycles per second in a human being with normal average hearing is about 76 decibels below one dyne per square centimeter, but in some people with exceptionally acute hearing the value may approach 85 These figures indicate that the acuity of persons possessing a high sensitivity of hearing closely approaches the thermal noise level, and a particularly good auditory system actually does approach this level Sivian and White s measurements indicate that even in the presence of greater auditory sensitivity in the spectrum between 1,000 and 6,000 cycles per second, the thermal noise in the air would limit the possible benefits Furthermore, it is not likely that animals possess greater acuity of hearing in this spectrum, as their hearing would also be limited by the thermal noises

In 1929 Bunch [16] classified audiograms for a large number of persons according to age groups Bunch s data are shown graphically in figure 6 The ordinates represent the hearing loss, in decibels in relation to the acuity of hearing of the 20 year old age group which is taken as zero decibels The abscissas indicate the frequency of the *applied tone* Bunch's data show that as one ages the acuity of hearing in the low frequency spectrum (up to about 500 cycles per second) is affected to a small degree and that it becomes progressively poorer with age as the frequency is increased above 500 cycles per second

Another series of audiograms was obtained by Montgomery [17] from persons ranging in age from 20 to 60 years Montgomery s data confirmed Bunch s observations however Bunch did not observe as marked a reduction in the higher frequencies with advancing age Montgomery s data showed that the average difference between the hearing of persons in the third and the sixth decades is approximately 7 decibels at 2,048 cycles per second and 23 decibels at 8 192 cycles per second

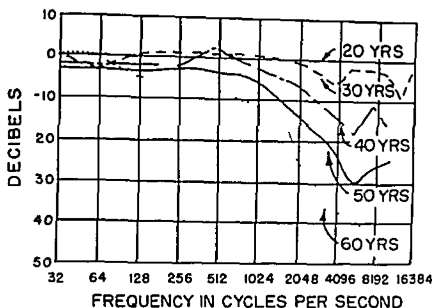


Fig 6 Average audiograms of persons at different age levels obtained by Bunch [16]

Audiometric tests were made on approximately 500,000 visitors to the Bell Systems Exhibits at the New York and the San Francisco World Fairs [18] The test frequencies were 440, 880, 1760, 3520, and 7,040 cycles per second It was apparent from these large scale tests that hearing acuity definitely diminishes with age as the frequency is increased beyond 1,000 cycles per second The average figures are closer to those of Bunch than to those obtained by Montgomery However, all of the tests show conclusively that a fortunate auscultatory condition exists for the average older physician, as cardiovascular and respiratory sound components of value are below 1,000 cycles per second in frequency

MINIMUM DISCERNIBLE CHANGES IN INTENSITY AND FREQUENCY

A law in psychology, known as the Weber Fechner law, states that the increase in stimulus required to produce a discernible increase in sensation maintains a ratio which is constant to the total stimulus Another way of stating the Weber Fechner law is that the magnitude of the produced sensation is proportional to the logarithm of the stimulus When the Weber Fechner law is applied to hearing, it may be stated that a barely perceptible increase in intensity of sound, as detected by the human ear, should exhibit itself as a constant which is independent of the sound intensity

Knudson [19] should be credited with the first accurate determination of the minimum perceptible variations in intensity between the frequency limits of 100 and 4,000 cycles per second Fletcher, by a modification of the testing procedure, extended the frequency spectrum between the limits of 35 and 10,000 cycles per second Knudson and Fletcher observed some varia

tion in the Weber Fechner law, as applied to hearing, in that the average ear was most sensitive to variations in intensity at sensation levels above 50 decibels and particularly did this hold true in the middle of the frequency spectrum An intensity variation of 5 to 10 per cent could be detected for sound levels above 50 decibels at approximately 2,000 cycles per second At the lower sound levels, and even more so at the extreme ends of the frequency spectrum, a decidedly greater percentage variation in intensity was required to produce a perceptible change As a comparative illustration, it was found that a 70 per cent variation in intensity was necessary at 2,000 cycles per second in order to produce the minimum perceptible change when the sound level was down to 10 decibels

In auscultation this characteristic of human hearing is an extremely important factor since the intensity level is most often 10 decibels or less, in addition the frequency spectrum which is involved is at the lower extreme As an illustration, at a frequency of 60 cycles per second a variation of 20 per cent is just perceptible when the sound level is 50 decibels, and a variation of as much as 200 to 300 per cent is necessary at the intensity level of 10 decibels

The human hearing mechanism is a far better detector of changes in frequency than it is of changes in intensity Shower and Biddulph [10] as well as Knudson [11] made extensive investigations to ascertain the minimum perceptible frequency changes at the various sensation levels They found that a higher sensation level of sound required less of a frequency change for minimum perceptibility Also the ear was found to be less sensitive to frequency variations as the lower end of the frequency spectrum was approached Between 500 and 4 000 cycles per second, the minimum perceptible fractional difference in frequency is 0 3 per cent at a *sensation level* * of 40 decibels At 64 cycles per second the minimum perceptible fractional difference in frequency is of the order of 1 1 per cent at 128 cycles per second it is 0 6 per cent and at 256 cycles per second, it is 0 4 per cent

AUDITORY LOUDNESS VERSUS STIMULATION

It is obvious that as the magnitude of the pressure wave which is producing auditory sensations is increased the perceived loudness is also increased However, it is most difficult to evaluate accurately the exact relationship between increase in sound pressure and increase in perceived loudness this problem has been a real challenge to psychophysicists, who have used many subjective approaches One procedure requires that the subject under test observe a tone at two levels of intensity and quantitatively compare the two in loudness Another procedure, known as the fractionation method uses a tone at a chosen level, and the subject under test operates

* The sensation level is the number of decibels the level of a tone is above its threshold of audibility for a particular subject or subjects

a control to vary the loudness by a designated amount such as one half of the original value Other methods require special assumptions, as for example, the indication of the midpoint of loudness between two tones, the balancing of one tone against the combination of two or more, the equating of a tone applied to one ear to the same tone applied to both ears

Fletcher and Munson [1] and Churcher,[2] using these various subjective methods, worked out a mean relationship between loudness and excitation pressures above threshold, which is graphically shown in figure 7 These

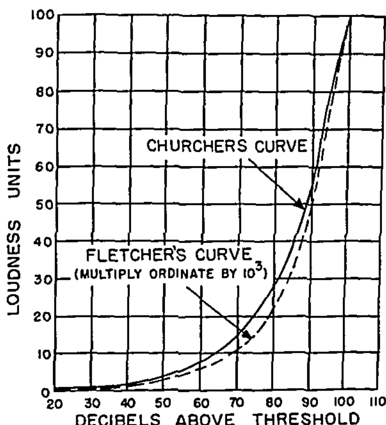


Fig 7 Perceived loudness in relation to the magnitude of the stimulus The arbitrary unit of loudness is derived by assuming that a tone with an intensity of 100 decibels above threshold has a loudness of 100 units

curves, although possibly somewhat crude because of the difficulty of expressing perceived sensation in exact numbers, may be used for approximate evaluations They indicate that a given logarithmic change in intensity is more effective at higher than at lower levels This means that an increase of one decibel in the level of an intense tone appears to add more to the perceived loudness than an increase of the same amount in a weak tone

The data of Fletcher and Munson and that of Churcher are not quite in accord with the Weber Fechner law (see p 28) If the curves of figure 7 conformed to this law, *they would be straight lines* Actually, figure 7 indicates that in auscultation the relationship is intermediate between linear

and logarithmic A linear curve which would fit at low intensity levels would rise too rapidly at the higher intensities a logarithmic curve that fits at the lower intensities would not rise at a sufficiently rapid rate at the higher intensities Experimental evidence is available which shows that the intensity function for cochlear response is linear up to the region of overloading, it would appear therefore that the effect of nonlinearity arises in the acoustic nerve[14]

MINIMAL TONAL PERCEPTION TIME

Another important measurement pertaining to auscultation is the minimum amount of time required for a tone to excite the auditory mechanism Stewart's[3] 'uncertainty principle' states that the ability of the ear to recognize a tone of brief duration decreases as the duration decreases Stewart expressed his principle mathematically by saying that the duration of the tone multiplied by the number of cycles of uncertainty in ascertaining the pitch of the signal equals unity It naturally follows from this expression that, in the lower frequency spectrum such as is encountered in auscultation, the frequency may be varied rapidly over a considerable portion of an octave without detection by the auditory *mechanism* This phenomenon may be illustrated if a vibrato with a frequency of 60 cycles per second is varied by *two semitones* at a rate of seven times per second, the variation cannot be detected by *the ear* *Table* 4 presents some probable values as regards tonal perception time

TABLE 4

SOME PROBABILITY TIMES IN TONAL PERCEPTION TIME

Cycles Per Second	*Weak Tones*		*Medium Tones*	
	Time (Seconds)	*Number of Cycles*	*Time (Seconds)*	*Number of Cycles*
128	0 0946	12 1		
256			0 06905	1, 6
384	0 062	24 08	0 0445	1 1
12	0 05 9	29 64	0 042,4	21 8

From Fletcher H

MASKING EFFECTS

Everyone has noted that in a noisy location he automatically increases the intensity of his voice to make *himself* heard The reduction in the ability of the ear to detect certain sounds in *the* presence of other sounds is technically known as masking Mayer[4] in 1876, was the first to show experimentally that low pitched sounds had a masking effect which differed from that produced by high pitched sounds Mayer claimed that a low pitched

tone was capable of completely masking a tone of higher pitch, but that a higher pitched tone was incapable of completely masking the lower pitched one

Because the equipment used by Mayer in his experiments was crude, the accuracy of his work was limited The Bell Telephone engineers continued the study and obtained more accurate data [8] They found that Mayer's conclusion regarding masking holds true only under certain conditions A low-pitched tone will not mask to any degree a high pitched tone far removed in frequency unless the low pitched tone is of a very considerable intensity, a higher pitched tone may easily mask a lower pitched tone if the frequencies are closely spaced

In a complex tone, for example, one consisting of three frequencies, such as 300, 400 and 2,000 cycles per second, with respective sensation levels of 50, 10, and 10 decibels, the experimenters found that the average human auditory system could detect only those tones having 400 or 2,000 cycles per second When the intensity of each component of the complex sound was increased by 30 decibels, so that the three tone levels were 80, 40, and 40 decibels, respectively, only the tones having 300 or 400 cycles per second were audible Under these conditions, when the 300 cycle per second tone was attenuated by only 8 decibels, it vanished completely From this example, it follows that the sensation produced on the auditory system by a complex sound is decidedly different in character, as well as in intensity, when the level is decreased or increased, even though no distortion is introduced *This peculiar characteristic of hearing follows the general rule that, as a complex sound becomes more intense, the low pitched tones become more prominent because the higher pitched tones are masked*

When a radio receiver is tuned to a station which is broadcasting a symphony orchestra, this masking effect is readily apparent if one listens first at high volume, and then turns the volume control to a much lower level At high volume the bass notes are reproduced with excellent fidelity, but at low volume the intensity of the bass notes is weakened more than that of the higher pitched notes

In auscultation this masking effect is even more noticeable because of the proximity of the frequency components which are involved It follows that obesity should produce an attenuating effect and cause a decided change in the quality of the heart sounds In fact, some of the important components of the heart sounds may be completely masked by obesity



AUDITORY FATIGUE

Another form of masking is introduced, especially in auscultation, when a sound of comparatively high intensity immediately precedes a sound of considerably lower intensity, for example, when a first or second sound precedes a murmur of low intensity The first or second sound of comparatively

great intensity has a tendency to fatigue the auditory mechanism of the observer temporarily, thereby masking the low intensity murmur The diaphragm type of stethoscope chest piece partially overcomes the masking effect by selective frequency attenuation, the principle of which will be discussed later Apparently, this auscultatory phenomenon is not appreciated by many physicians, as many use either the open bell stethoscope or the diaphragm stethoscope exclusively

VARIATION OF LOUDNESS WITH FREQUENCY

Because of the fact that the human auditory mechanism does not respond equally to different pitches, tones of the same intensity but of different frequencies produce different sensations of loudness Also, if the intensities of the tones of different pitch but of equal loudness are increased by an equal amount, an unequal sensation of loudness is produced Kingsbury and, more recently, Fletcher and Munson [1] obtained quantitative measurements on the determination of equal loudness curves for average persons

Figure 8 shows the curves obtained by Fletcher and Munson [1] The abscissa of the graph, which is a logarithmic scale, represents the frequency *spectrum* in cycles per second that is covered by the average normal human auditory system, and the ordinates are the sound *pressures* or intensity levels, before entry into the ear, in decibels above threshold The pressure measurement external *to the* ear is known in the science of acoustics as a free field measurement, the pressure is measured at a location free from bounding surfaces and *then the* ear is placed at the same location where the pressure measurement was made The sound pressure levels in decibels along the axis of ordinates are referred to 0 0002 dyne per square centimeter, which is the generally accepted value for the threshold of audibility of normal human auditory systems

The family of curves in figure 8 was obtained for equal loudness contours which are measured on the graph *in phons* A phon is an acoustical unit for measuring the loudness of a tone The number of phons is equal to the number of decibels a 1,000 cycle per second tone is above the reference sound pressure when it is subjectively judged to be equal in perceived loudness to the tone in question From the family of curves of figure 8, it may be seen that the maximum sensitivity of the average human auditory system is in the vicinity of 3,000 cycles per second In the frequency spectrum beyond 1,000 cycles per second, a given *increase* in the intensity of stimulation, as measured on the axis of ordinates, results in approximately the same *increase* in loudness whereas at lower frequencies the same increase in stimulation produces a much greater increase in loudness Resolving this hearing characteristic into actual values, at a frequency of 600 *cycles* per second, a sound pressure *increase* of 76 decibels is necessary to create a loudness level of 80 phons above the threshold of audibility level of zero phons, whereas at

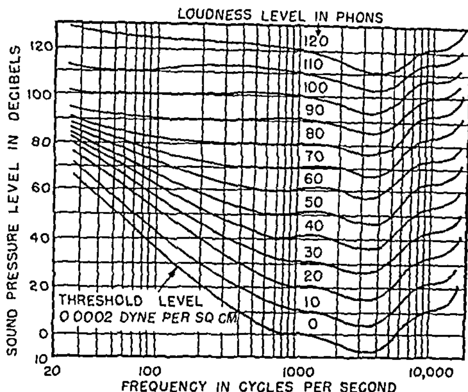


Fig 8 Relative sensitivity curves of the average normal human auditory system where the ordinate is the sound pressure level measured in a free field before entry into the subject's ear (From Fletcher H and Munson W A)[21]

100 cycles per second, an increase of 43 decibels of sound pressure is required to produce an equal increase, at 8,000 cycles per second, 76 decibels are required, and at 1,000 cycles per second, 80 decibels It is important that this *nonlinear characteristic of hearing be taken into consideration* when designing a stethoscope, for a modification of the frequency response characteristic *of the stethoscope will seem to alter the quality of the auscultatory sounds*

In 1940 Stevens and Volkman[21] obtained another family of loudness contour curves, shown in figure 9, which differ somewhat from those of Fletcher and Munson[21] The graph of Stevens and Volkman expresses the sound pressure level at the eardrum, whereas Fletcher and Munson measured the sound pressure in a free field external to the ear *It is generally* observed that if sound pressure is measured at the eardrum, 6 to 10 decibels more are required to produce audibility than are required if external free field measurement is used *A difference in loudness level of approximately* seven decibels between Fletcher and Munson's conditions of excitation and *those of Stevens and Volkman, is evident from figures 8 and 9*

MONAURAL VERSUS BINAURAL HEARING

Prior to Fletcher's experiments on hearing, it had been believed that the loudness of perceived sound depends on the *number of nervous impulses*

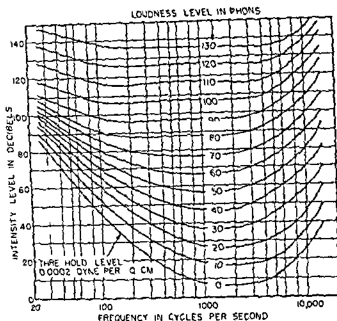


Fig 9 Relative sensitivity curves of the average normal human auditory system where the ordinate of the graph is the sound pressure level measured at the eardrum (From Ste ens S S and Volkman J)[26]

reaching the brain According to this theory, bmaural listening should pro duce twice the loudness of monaural listening Fletcher applied alternately to each ear a tone which produced equal perceived loudness, then he ap plied the tone simultaneously to both ears He observed that with binaural hearing he had to reduce the sound by *a* decibels to make the sound as loud as it had been with monaural listening and that the value of *a* was different at various sensation levels The average readings obtained from a group are given in table 5 (see discussion of respective merits of binaural versus monaural auscultation by stethoscopic and direct means, (pp 49 50)

From Fletcher s data it appears that when both ears are stimulated iden tically the effects from both ears are brought together in the central nervous

TABLE 5

DEVIATIONS IN THE INDIVIDUAL PHYSICIAN S HEARING FROM THE AVERAGE NORMAL THRESHOLD CURVE OF AUDIBILITY

Sensation Level	*a in Decibels*	*Increase in Loudness*
29	3 1	1 43 Times
46	5 3	1 84
64	10 0	3 16
81	9 0	2 82
99	8 2	2 57

From Fletcher H

processes and effectually fused When a phase difference is present in the tones reaching both ears, the fusion in the central nervous processes is incomplete and the auditory phenomenon of sound localization or stereophonic effect is produced [14] This form of auscultation has been used by several investigators for the localization of cardiovascular sounds and murmurs (see p 40)

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4 / Description of Heart Sounds Murmurs and Noncardiac Chest Sounds from a Physical Point of View

TRANSMISSION CHARACTERISTICS

Acoustics, as related to auscultation, deals with the vibrations or disturbances set up in the chest, with their transmission through the component sections of the chest, as well as through the stethoscope, and with the resulting effects on the observer's auditory mechanism Sound, which is the result of vibration of a medium, is capable of traveling through a solid, liquid, or gas as a compressional wave, unless the medium is entirely inelastic In other words, the ability of sound to travel through matter depends on the elasticity, viscosity, and density of the medium If the effects of viscosity are small, as is the case with water, air, metal, and bone, the sound energy may be transmitted with little loss In some media, such as soft rubber or fatty breast tissue, the sound waves are almost immediately suppressed

In a homogeneous substance a sound wave propagates itself at a velocity the magnitude of which depends on the physical properties of the medium, and the attenuation of the wave is governed by the viscosity and the spreading of the sound energy over a larger surface as the wave progresses When sound energy travels from one medium into another of different physical properties or when there is no longer homogeneity of medium, transmission losses in the form of refraction and reflection take place Some media are capable of transmitting low frequency vibrations with less attenuation than high frequencies, and vice versa, this results in distortion of the sound In the human body there are many paths along which heart and chest sounds travel to reach the surface A large percentage of the sound energy never reaches the surface because of losses due to viscosity, elasticity, density, spreading reflection, and refraction Naturally, the intensity of the sound is maximum over that portion of the chest where the sound pursues a path of minimum attenuation, this is the location the physician normally selects with his stethoscope Auscultatory experience has shown that murmurs produced by lesions of the four valves of the heart are heard with maximum intensity in certain areas of the chest, although there may be other sections of the chest surface which are closer to these valves

CLASSIFICATION OF SOUNDS

Various studies have been made to ascertain what frequency band is involved in auscultation[1-4] Cabot and Dodge[5] came to the following conclusions

> Practically all the sounds of interest in auscultation are made up of frequencies below 1,000 cycles per second In general the upper and lower frequency limits for the pathologic sounds in any particular case are not sharply defined
>
> Presystolic murmurs as a class are characterized by a greater predominance of low frequencies than other murmurs, and are almost invariably termed 'low pitched'
>
> The frequency bands of importance in systolic and diastolic murmurs are broadly the same In each group extremely low pitched and high pitched cases are found but there appears to be nothing in the quality of the sounds that is characteristic of murmurs occurring in systole or diastole In our limited studies, we have not been able to associate a particular frequency band with murmurs produced by a lesion of a given kind
>
> Lung sounds as a class have a proportionately smaller amount of low frequency components than heart sounds The descriptive words 'coarse and fine' as applied to râles are usually associated with conspicuous low and high frequency components Breath sounds are, broadly, higher pitched than most heart sounds as evidenced by the scarcity of components below 240 cycles per second

Williams and Dodge[2] observed the distribution of energy in normal heart sounds (table 6) Above 110 cycles per second, the energy components of

TABLE 6

ENERGY CONTENT IN NORMAL HEART SOUNDS

Frequency in Cycles per Second	*Energy in Per Cent*
50 60	56
60 70	27
70-80	10
80 90	4
90 100	2
100 110	1

From Williams H B and Dodge H F[2]

the normal heart sounds were negligible They further observed that low pitched heart murmurs have frequencies lower than 400 cycles per second and that high pitched murmurs range from 120 to 660 cycles per second The frequencies of systolic and diastolic murmurs range between 120 and 660, occasionally ascending to 1,000 cycles per second Cabot and Dodge[5] noted that, for the most part, the frequencies of presystolic murmurs lie below 140 cycles per second, but these murmurs contain components up to 400 cycles per second They stated that the pericardial rub is composed of frequencies between 140 and 660 cycles per second, râles, between 120 and

1,000, amphoric breathing, between 240 and 660, and bronchial breathing, between 240 and 1,000 cycles per second

In our observations of the various heart sounds and murmurs, we have not encountered any frequency components of noticeable value above 650 cycles per second However, we have encountered frequency components of heart sounds that were well below the range of the human ear (obtained by recording the sounds graphically) Exactly where the lower limit of the sounds occurred was difficult to observe, for they were intermingled with the low frequency waves caused by motions of the chest wall The lowest frequency limit of heart sound components is in the vicinity of 5 to 10 cycles per second, although sounds with a frequency of less than 30 to 40 cycles per second cannot be heard if the intensity is low

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5 / The Acoustic Stethoscope ✓

GENERAL CLASSIFICATION

Acoustic stethoscopes may be classified as monaural, binaural, and differential The monaural stethoscope described earlier (p 13) was invented by Laennec and later improved by Piorry It is still in use on the continent of Europe, but the binaural stethoscope is more commonly used There are many kinds of binaural stethoscopes, but the only two important types are those with open and diaphragm chest pieces

Differential stethoscopes are used primarily for localizing and comparing sounds Their operation depends on the well known characteristic of hearing termed "auditory localization," or the ability to ascertain the direction of a sound source by means of phase differences Phase difference depends on when the sound reaches each ear, and, in turn, registers on the brain In addition, there is a stereophonic effect which gives the sound characteristic depth and extensiveness

Alison,[1] in 1859, described a differential stethoscope which consisted of two monaural stethoscopes of the flexible tube type Muralt, in 1910, devised a differential stethoscope which allowed him to listen simultaneously to two lung areas Muralt's stethoscope incorporated two similar chest pieces, so arranged that two tubes of equal length connected each chest piece with each ear four tubes were used in all In 1934, Froschels[3] suggested using a differential stethoscope to study the sounds produced by the vocal cords to detect laryngeal paralysis Froschels' stethoscope was a modification of Muralt's, in that it consisted of two similar chest pieces and two tubes passing through an X which allowed an ipsilateral or contralateral course of sound to each ear Hawthorne,[4] in 1935, described a differential (double) stethoscope which was similar to Alison's In 1936, Nicolai[5] described what he termed a "stereostethoscope," to detect differences in sounds originating in the two mandibular joints The Nicolai stereostethoscope was identical with the one described by Alison Nicolai and Hintschmann[6] found it useful in the study of pulmonary and cardiac disease

In 1937, Kerr and his associates[7] described an instrument which they called the "symballaphone" and which they classified as a "modified stethoscope for the lateralization and comparison of sounds" The symballaphone was similar to Muralt's stethoscope, except that a longer connecting tube

from each chest piece to the opposite ear piece was employed in place of the direct tube to the ear piece on each side

Prior to 1940, many variations and styles of bells had been manufactured and marketed, with wide claims for their performance, but rarely had a theoretical or scientific explanation of the acoustics of the bell been attempted Similarly, the acoustics of the stethoscope structure between the bell and the ear had been ignored In 1940 Johnston and Kline[8] presented their data on the acoustic stethoscope The testing procedure they employed was significant, in that it was one of the first attempts at a quantitative analysis of stethoscope performance

The experimental setup was composed of

> A special telephone receiver driven by a variable frequency oscillator placed within the heart of a cadaver by means of an abdominal-diaphragmatic approach so that vibrations of any desired amplitude and pitch were transmitted to the intact thoracic wall over the heart The vibrations were picked up by the end pieces of the stethoscope and conveyed through the columns of air of the instrument to a condenser microphone The output from the microphone was passed into a suitable vacuum tube amplifier and thence to an output meter of the rectifier type The readings of this meter were proportional to the intensity of the waves striking the diaphragm of the condenser microphone All tests were made with the end pieces of the stethoscope held rigidly against the same point on the precordium and great care was taken to be sure that each end piece was in air tight contact with the skin

The Johnston and Kline calibration procedure was as follows

> Before any tests on stethoscope units were undertaken attempts were made to standardize the vibrations transmitted to the wall of the chest so that their amplitude would be constant over the entire range of frequencies employed This problem proved to be difficult and was not solved to our satisfaction We finally used a crystal vibration pick up fixed at the chosen point on the precordium and determined the value of the resistance which would give the same response on the output meter at each frequency

In 1941 the experimental data and acoustical theory on stethoscope performance described by two of us (M B R and H B S)[9] showed a number of flaws in Johnston and Kline s experiments It is interesting that although considerable data have been presented for improving stethoscope performance since 1941 the average stethoscope used today (1960) is still lacking in many elements of scientific design and has been changed little in the last thirty years However the stethoscope designed by the two authors mentioned above combines the results of research on the physics and acoustics of the acoustic stethoscope in a single instrument with as nearly ideal sound transmission for cardiac study as appears presently possible

PRINCIPLE OF OPERATION

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PRINCIPLE OF OPERATION

The stethoscope is a device which provides a closed acoustic system for conducting sounds that originate in the body of the patient to the observer's

ear The sound transmission medium is a closed column of air in the case of the binaural stethoscope, which employs rubber tubing as a link between the chest piece, or sound accumulator, and the ear pieces In the rigid monaural stethoscope, the sound travels by both bone conduction and air column transmission The bone conduction is dependent on contact with the ear as well as with the material of which the stethoscope is made, whereas transmission by air column depends on the pressure changes that take place

THE OPEN BELL

For the most part, in medical literature the bell portion of the stethoscope has been regarded merely as an accumulator or collector of sounds The general trend of thought has been that, if the sound as it accumulates on a patient's chest could be gathered from the largest practical area, there would be a proportional increase in intensity when it is transmitted to the ear [10,11] According to this reasoning, the larger the contact area of the bell consistent with good seating, the greater the heart sound intensity Limited clinical trial seemed to substantiate this supposition Various types of bells, the construction of which was based on these assumptions, have been marketed Other designers have gone a step further by giving the internal portion of the bell various geometric shapes to aid the accumulating properties

It is our opinion that the open bell should not be considered primarily as an accumulator When it is held in the open, the open bell exhibits acoustic characteristics that are entirely different from those which it exhibits when it is applied to the chest *When an open bell is applied to the chest, the skin bounded by the lip of the bell forms a diaphragm, and the fleshy portion under the skin acts as a damping medium* In other words, the condition is similar to that encountered in a microphone or telephone receiver

The physical principles governing the operation of a damped diaphragm are well known and may be found in textbooks on telephony Any diaphragm has a natural frequency of vibration, or resonance point, which is dependent on its inherent inertia, elasticity, diameter, and tautness The maximum sensitivity of the diaphragm to external excitation occurs at the point of resonance In other words, if a diaphragm (made of any material) is excited by sound or vibrational energy, the pitch or frequency of which is varied from zero to infinity while the amplitude is kept constant, the diaphragm will oscillate in unison with the exciting medium and reach a maximum oscillation amplitude at its point of resonance The following are some of the general rules governing the action of diaphragms

1 The more taut a diaphragm is drawn the higher its natural frequency of oscillation becomes

2 The larger the diameter of the diaphragm the lower is its natural frequency

3 Damping suppresses the maximum vibrations of the diaphragm that occur at the resonance point

4 When the natural frequency of a diaphragm is increased the upper frequency range

to which the diaphragm is capable of responding is increased However this effect cannot be attained without lowering the sensitivity of the diaphragm throughout its entire frequency range Thus a diaphragm with a higher natural frequency is less sensitive to the lower frequencies or pitches than a diaphragm of lower natural frequency

5 Diaphragms have harmonic resonance points which occur at multiple frequencies of the fundamental In auscultation we are concerned only with the fundamental

6 At the frequency of mechanical resonance of the diaphragm the effects of the inertia and elasticity of the diaphragm balance each other and its velocity is in phase with the impelling force

If the open bell of the stethoscope is regarded as a device for producing a diaphragm effect when it is applied to a patient's chest and if the physical characteristics of diaphragms are kept in mind, many clinical observations can be explained It is a commonly observed fact in auscultation that, for a bell of a given size, the greater the pressure with which the bell is applied to the patient's chest, the less is the apparent intensity of the first and second heart sounds and the higher is the pitch This phenomenon can be readily explained by the fact that, with greater pressure, the skin bounded by the lip of the bell is drawn more taut, this produces a diaphragm with a higher natural frequency than that produced when the pressure is light As has been said, a diaphragm with a high natural frequency extends the upper portion of the frequency band, but, at the same time, it attenuates the over all level except at the point of resonance It is to be expected that a diaphragm with a high natural frequency would attenuate the lower frequency components of the heart sounds and tend to bring out the higher frequency components, thereby altering the pitch and reducing the intensity of the first and second heart sounds in which the low frequencies predominate

Normally, the smaller the diameter of the opening of the bell the higher is the pitch of the sounds This characteristic is explained by the fact that with a small bell, the diaphragm dimensions are small, this tends to give it a higher natural frequency Also, in order to obtain a reasonably good seal with the skin, sufficient pressure must be exerted to stretch the skin enclosed by the lip of the bell, thereby additionally raising the natural frequency With a bell of larger diameter the tendency to stretch the skin is proportionally reduced, this makes a stethoscope with a bell of large diameter much less sensitive to pressure variations as regards pitch than a stethoscope with a small bell

The pressure pitch characteristic of stethoscopes has a useful application in auscultation when a bell of normal size (approximately one inch in diameter) is employed By varying the pressure on the patient's chest, the physician can create a variable filtering action on the heart sounds that are transmitted by the closed system In order to clarify the principle, let us consider a pathologic condition in which there are loud first and second heart sounds a loud systolic murmur of fairly low pitch, and a very low intensity,

hardly audible, high pitched diastolic murmur Because of the comparatively great intensity of the sounds other than the diastolic murmur, the latter is masked (see pp 31 32) When the pressure with which the stethoscope is applied is increased, the natural frequency of the diaphragm formed by the skin is raised, thereby attenuating the lower pitched components, of which the first and second heart sounds, as well as the systolic murmur are composed, and allowing the higher pitched diastolic murmur to stand out better as a result of the decreased masking effect When the pressure with the stethoscope is decreased, low pitched murmurs, third heart sounds, and gallop rhythms may be brought out more distinctly

Another important consideration in regard to the open bell is the effect of its internal dimensions and shape As has been mentioned previously, various geometric shapes and forms have been devised to improve the accumulating properties of the open bell, with a resulting loss of accumulation in most cases The fallacy in making bells of certain geometric shapes becomes obvious if we stop to consider that the pressure variations at the ear which are produced by the skin diaphragm in the stethoscope proper are inversely proportional to the volume of the bell Thus, an infinitely small volume produces a maximum variation in pressure, which, in turn, manifests itself as a sound of maximum intensity The only important consideration in designing a bell, aside from keeping the internal volume at a minimum, is to have it so shaped that, in the case of an obese patient, the bell will not fill with flesh to such an extent as to decrease the diameter of the diaphragm and, therefore, its effect

At this point it is logical to consider the relationship of bell resonance to dimensions A stethoscope bell may be placed in the same class as the Helmholz resonators, an analysis of which may be found in almost any text on acoustics Although it is somewhat beyond the scope of this discussion to go into the acoustics of Helmholz resonators as applied to stethoscope bells, it is of interest to note that a bell with an internal lip diameter of 5 8 cm and an internal body depth of 0 9 cm, with a 0 6 cm hole 1 cm long, through the threaded portion (which attaches to the stethoscope proper), has a theoretical resonance frequency of approximately 500 cycles per second Because of the fact that so large a bell has a resonance frequency which lies at about the upper limits of heart and chest sound frequencies, and because bells of smaller size exhibit proportionally higher resonance frequencies, *it is impossible to obtain any really effective filtering by bell shape modifications other than those previously mentioned*

THE DIAPHRAGM CHEST PIECE

The diaphragm chest piece (Bowles type), which is commonly employed in auscultation, is especially useful in detecting faint, high pitched sounds, such as the barely audible, high pitched, diastolic murmur of aortic insufficiency and the high pitched "bronchial" respiratory sounds Essentially,

the principle of its operation is similar to that of the open bell when it is applied to the patient's chest, except that additional attenuation of the lower pitched heart and chest sound components is obtainable Of course, the smaller the diameter of the open bell, and the greater the pressure with which the bell is applied, the greater the low frequency attenuation, but, to obtain an equivalent degree of attenuation with the open bell, the pressure would be great enough to hurt the patient By interposing a diaphragm of bakelite, or any other plastic whose natural frequency is in the desired range, the necessary attenuation can be obtained with light pressure on the patient s chest In other words, the plastic diaphragm becomes a substitute for the skin diaphragm, and the flesh of the chest acts as a damping medium

All of the general principles which are applicable to the skin diaphragm apply equally well to the plastic diaphragm It is well to mention at this time that the plastic diaphragm is referred to because it is the one most generally employed The theory of operation of a metal diaphragm is iden tical, but a foreign, metallic ring may be superimposed upon the sounds

As is the case with the open bell, for maximum efficiency, the volume of air in the diaphragm chest piece should be as small as possible The internal volume of a diaphragm bell may be considerably less than that of an open bell of similar diameter, for the stiff diaphragm prevents the tissue of the patient's chest from entering and decreasing the effective diameter

OPEN BELL VERSUS DIAPHRAGM CHEST PIECE

An attempt was made to verify experimentally the theory of the stetho scope bell which has been discussed A frequency characteristic test was made using the four chest pieces shown in figure 10 Figure 11 is a schematic sketch of the apparatus employed in the experiment

The posterior portion of the patient's chest was allowed to rest against a loud speaker, with its associated baffle Throughout the entire test the patient was kept in one position relative to the loud speaker The four chest pieces shown in figure 10 (the chest pieces may be screwed into the specially designed microphone, the characteristics of which will be discussed later,



Fig 10 Chest p e es employed in the ve ification of the stethoscope bell theo y (F om Rappaport M B and Sprague H B) 9

see pp 65, 66) were placed one at a time on an area marked on the front of the patient's chest In other words, the adjustable frequency sound energy was fed into the patient's chest and picked up by the microphone and its chest pieces, it was then amplified and fed into a cathode ray oscillograph, used as a recorder Equal pressure on the patient's chest was used with all four chest pieces

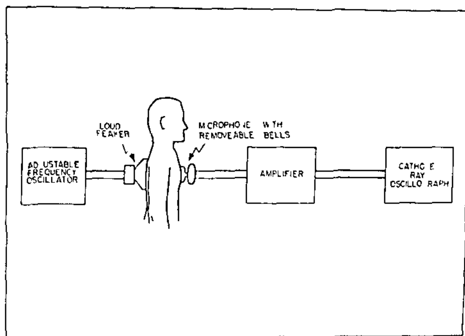


Fig 11 Schematic diagram of apparatus employed in obtaining the curves shown in figure 12 (From Rappaport M B and Sprague H B)[9]

A frequency response curve was recorded for each chest piece The curves thus obtained had practically no meaning, for they included the characteristics of the loud speaker and the transmissional mediums of the chest However, subtraction of one response curve from the other canceled the loud speaker and chest transmissional characteristics, thus allowing direct comparison of the respective chest pieces For our purpose, a comparison of the diaphragm chest piece with the open bells (using the diaphragm chest piece as a reference value) supplied useful information The resultant curves of the three open bells and the diaphragm chest piece are compared in figure 12, the reference value was zero decibels

The dimensions of the four bells were as follows The lip diameter of the large bell was 2 0 in, of the medium bell, 1 5 in, and of the small bell, 1 0 in The diaphragm bell was a typical Bowles chest piece, with a diaphragm 0 015 in thick and a freely working diameter of 1¾ in The internal volume of the large bell was 12 7 c c, of the medium bell, 6 2 c c of the small bell, 2 3 c c, and of the diaphragm bell, 2 5 c c

The following are some of the more important general conclusions that may safely be drawn from the graph of figure 12

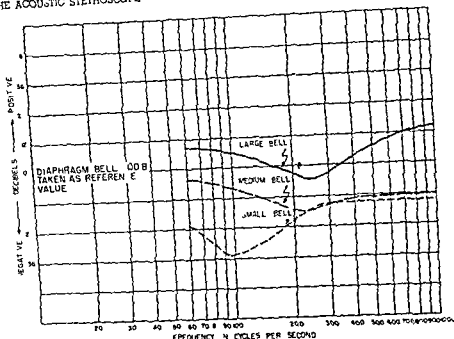


Fig 12 Curves of the three open chest pieces compared with the diaphragm chest piece The higher the decibel reading the higher the relative efficiency of the chest piece (From Rappaport M B and Sprague H B)[19]

1 The efficiency of the three open bells improves more than that of the diaphragm chest piece as the frequency is decreased

2 The larger the diameter of the open bell the more efficient is the chest piece at the lower frequencies

3 The large bell exhibits a resonance effect in the upper auscultatory region that is characteristic of a Helmholz resonator of such dimensions The resonance points of the smaller bells are above the auscultatory range and therefore are *not* shown in the graph

It has been mentioned previously that a diaphragm bell is useful in bringing out certain high pitched murmurs because it suppresses the lower frequency heart sound components that tend to mask the low intensity murmur of high pitch The commonly used diaphragm chest piece, such as the Bowles type shown in figure 10 employs a bakelite diaphragm 0 015 in thick, with a freely working diameter of 1⅜ in This diaphragm was selected because clinical trial showed that it suppresses the lower pitches sufficiently to bring out the higher pitched murmurs that are normally masked Thicker diaphragms or diaphragms with a higher natural frequency are not normally used because they produce such a pronounced over-all attenuation of the heart sounds that they are more of a disadvantage than an advantage If the efficiency of the acoustic stethoscope could be increased enough to overcome the general reduction of sound intensity which occurs when thicker diaphragms are employed the hard to hear, high pitched diastolic murmurs might be more easily detected This proved to be true when an amplifying

stethoscope was employed, a diaphragm chest piece with a higher natural frequency and an adjustable volume control was used This test will be discussed in greater detail later (see p 86)

Figure 13 shows two records which were obtained from a patient with an aortic systolic and diastolic murmur The filtering action, or attenuation, of the low frequency components of the murmurs, as well as of the second heart sound (the only one present), is obvious Systole and diastole may be timed accurately on the phonocardiogram by recording the electrocardiogram simultaneously The nature of the mechanism producing the records will be discussed later (see p 86)

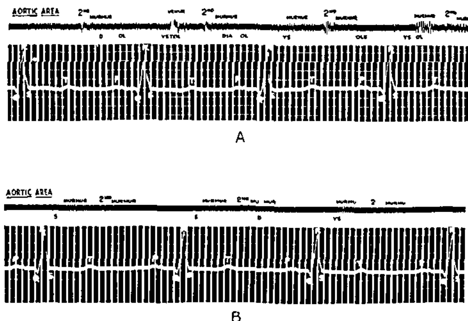


Fig 13 Graphic records (phonocardiograms) of an aortic systolic and diastolic murmur A taken with the large open chest piece shown in figure 10 B taken with the diaphragm (Bowles type) chest piece shown in figure 10 (From Rappaport M B and Sprague H B)[9]

BINAURAL EFFECTS

Figure 14 is a schematic diagram of apparatus employed to ascertain the effect of a binaural system in a stethoscope An extremely quiet room was selected for the test (noise would affect the accuracy of the test because it would alter the threshold of hearing) The apparatus consisted of an adjustable frequency oscillator which was capable of producing a sinusoidal electrical wave practically free of any harmonic content The frequency was adjustable over the auscultatory range The electrical energy output at the various frequency settings was adjustable by means of a continuously variable control The measured sinusoidal electrical waves were fed into a phone which could be placed to the ear of the person being tested, or led to the ears through a binaural attachment such as that used on stethoscopes

The phone was applied to the ear with constant pressure throughout the

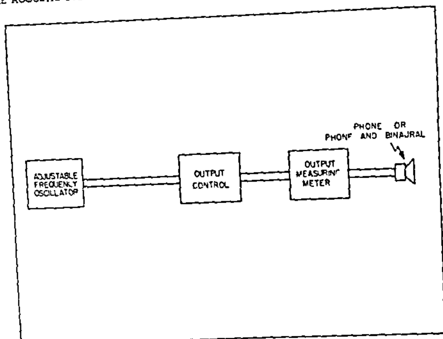


Fig 14 Schematic diagram of apparatus employed in determining the binaural effect (From Rappaport M B and Sprague H B)[19]

test For each frequency setting throughout the auscultatory range, the person who was being tested adjusted the intensity of the tone to the point where the tone was just audible (threshold level), and the energy entering the phone was then measured The same procedure was followed for the same phone with the binaural attachment

If the resultant data were plotted in decibels against frequency in cycles per second the characteristics of the phone would have to be taken into consideration but if the phone data are subtracted from the binaural data at each frequency tested the characteristics of the phone are canceled and a direct comparison of hearing by the two systems is obtained The reference level in figure 15 was taken as zero decibels so that if any portion of the curve showed a positive decibel reading it was an indication that the binaural system was more efficient by whatever the decibel reading happened to be when the curve was negative the monaural system was more efficient

The following are some of the important relationships that exist between the monaural and binaural systems in auscultation

1 Direct telephone receiver application may be considered similar to direct auscultation

2 A binaural system introduces resonance peaks in the auscultatory spectrum as may be seen in figure 15

3 In the range from 60 to 400 cycles per second which includes the greater part of the auscultatory range the binaural system is on an average 20 decibels better than the

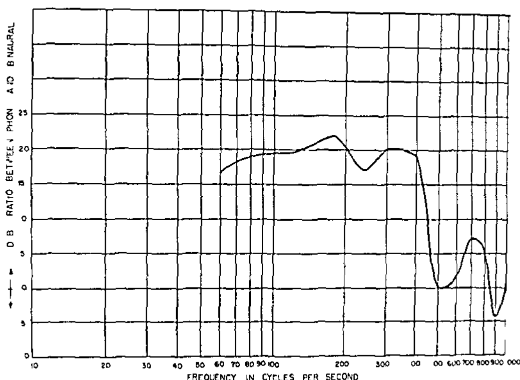


Fig 15 Relationship between monaural and binaural hearing in auscultation Monaural hearing may be considered the equivalent of direct auscultation whereas binaural hearing takes in the additive effect of both ears and the effects of the binaural stethoscope attachment (From Rappaport M B and Sprague H B)[9]

monaural, this is equivalent to a ten fold increase in sound pressure at the eardrum at the threshold level A ten fold increase in sound pressure at the eardrum may mean the difference between making a threshold murmur audible and not hearing it at all

4 Only between 850 and 1 000 cycles per second is monaural or direct auscultation more efficient than binaural and this range is too high to be useful in cardiovascular auscultation

Effects of tubing dimensions

An important consideration pertaining to stethoscope performance is the length and caliber of the rubber tubing It has been mentioned previously that the pressure variations at the ear, produced by the motion of the skin or plastic diaphragm, are inversely proportional to the internal volume of the stethoscope, so that an infinitely small volume would theoretically exhibit a maximum variation in pressure, which, in turn, would manifest itself as a sound of maximum intensity Obviously, therefore, the tubing should be as short as possible, and the caliber as small as possible The wall of the tubing should be sufficiently rigid for maximum efficiency, any wall motion reduces the effective pressure variation transmitted to the observer s ear The metal binaural tubing of the stethoscope must be considered as an extension of the rubber tubing, and its effects on the over all performance of the stethoscope must also be included

Length In 1941, two of us (M B R and H B S)[9] performed a test to

ascertain the actual change in efficiency of a binaural stethoscope, caused by lengthening the rubber tubing The apparatus employed was identical with that shown in figure 14 *The condition* under which the test was made were the same as those for the binaural effect experiment The only difference in the entire test was that a threshold of hearing curve (plotted in decibels of input to the telephone receiver to produce the *threshold of hearing* level against frequency in cycles per second) was obtained for the phone and metal binaural attachment for various lengths of tubing Two tubes were used to connect the binaural to the phone (audiophone, see p 53) *Figure* 16 shows the curves obtained with three sets of tubing, which were 26 12 and 3 inches long respectively The added length of the metal tube of the binaural was 9 inches for each ear

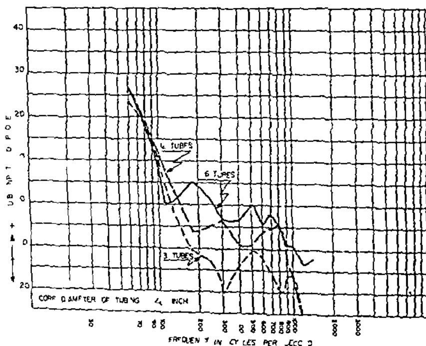


Fig 16 Curves illustrating transmissional losses in a binaural stethoscope as a result of increased length of the tubing (From Rappaport M B and Sprague H B)

The following are the general conclusions that may be drawn from a comparison of the three curves in figure 16

1 Below 100 cycles per second, the efficiency is not affected materially by the length of the tubing

2 From 100 to 1 000 cycles per second the length of the tubing exerts a considerable effect on the efficiency that is *the efficiency decreases as the length of the tubing increases*

The changes in the efficiency of an acoustic stethoscope, which are caused by varying the length of the tubing, although they are *not* considered by

most stethoscope users, produce a marked effect on the loudness of medium pitched and high pitched murmurs For example, a medium pitched murmur frequency component of 200 cycles per second would be attenuated 15 decibels by substituting 26 inch long commercial stethoscope tubing, with a caliber of $\frac{1}{16}$ in for similar rubber tubing 3 inches long This means that the substitution of the 26 inch rubber tubing for the 3 inch tubing reduces the sound pressure at the ears 5 6 times, at 300 cycles per second the attenuation is about the same A high frequency murmur component, such as 400 cycles per second, is attenuated about 10 decibels or about 3 1 times, at 500, the attenuation is about the same, at 600, it is attenuated about 15 decibels or about 5 6 times, and at 700, 13 decibels, or about 4 5 times This attenuation occurs in the region where the low intensity, medium pitched and high pitched diastolic murmurs are present at about the threshold of audibility, and every possible increase in stethoscope efficiency in this portion of the auscultatory spectrum is important

It is obvious that a stethoscope with 3 inch rubber tubes is too short for optimal handling in auscultation, 10 inch rubber tubes have proved satisfactory in the usual forms of manipulation although some efficiency is sacrificed

BORE As mentioned previously, the frictional resistance offered to the column of air by the walls of the tubing affects the over all efficiency of the stethoscope That is, the efficiency of a stethoscope decreases as the resistance to the transmission of pressure variations increases, the resistance is increased as the caliber is decreased In eliminating the resistance component, it still must be remembered that the greater the volume, the less the efficiency Therefore, the most efficient tubing for a stethoscope should be as short as possible and compromise on the resistance or caliber

Due to the lack of suitable instrumentation at the time our initial experiments were made (1939 1941), exact determinations were not made with regard to tubing bore [9] During World War II, a sound pressure meter [1-] was devised by the Research on Sound Control group at Cruft Laboratory, Harvard University One of us (M B R) collaborated on the project of making this instrument practical and reproducible With the aid of this sound pressure meter and several other devices, we were able to make finite measurements pertaining to the caliber of stethoscope tubing [13]

Figure 17 is a photograph of the instrumentation used for the determination of the effects of tubing caliber on acoustical stethoscope efficiency Basically a sound pressure meter is an instrument, or combination of instruments, comprising a microphone, an amplifier, and an indicating meter, with a flat or uniform response as a function of frequency in the desired frequency spectrum It indicates the value of the sound pressure in dynes per square centimeter or decibels above a reference, such as the average human threshold of hearing The commonly used threshold reference is equal to 0 0002 dynes per square centimeter

When making a sound pressure determination, the adjustable frequency

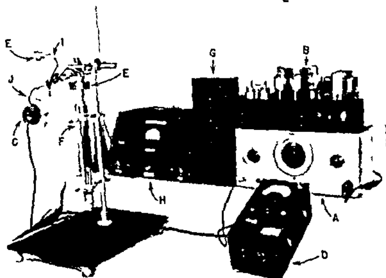


Fig 17 Instrumentation for determining the effects of tubing caliber on acoustical stethoscope efficiency The components are A adjustable frequency oscillator B amplifier C calibrated phone D vacuum tube voltmeter E artificial ears F sound pressure meter G decade precision resistance box H sound pressure meter control box I stethoscope binaurals and J stethoscope tubing (From Rappaport M B and Sprague H B)[13]

oscillator A, is set at a predetermined frequency (Fig 17) By means of the oscillator intensity control and the amplifier B just above it, a sinusoidal electrical signal is applied to the calibrated audiophone, C The intensity of the electrical signal which enters the audiophone is monitored by means of the vacuum tube voltmeter D The audiophone transforms the electrical signal into an equivalent sound which is conducted by the stethoscope tubing to the artificial ears E An artificial ear[14 1] is an acoustical cavity whose acoustical impedance is intended to simulate the impedance of the average human ear and thus make possible sound pressure measurements as they would occur in the ear Figure 18 shows a close up view of the artificial ears used The spiral like tube visible on the artificial ear to the right is a high pass filter which simulates the action of the eustachian tube and prevents a build up of constant pressures on the sound pressure meter, F shown in figure 17 The sound pressure meter in turn measures the pressure of the sound at the ear pieces of the binaurals in dynes per square centimeter or decibels above threshold The decade box G between the oscillator and sound pressure control box H, is for the purpose of matching electrical impedance so that constant power at varying frequencies is delivered by the audiophone to the tubing

The binaurals used in this investigation were of the common commercial

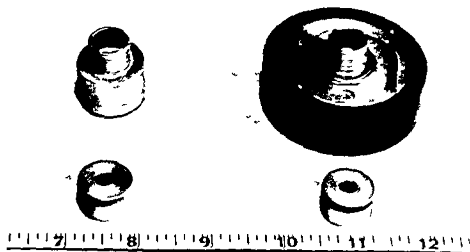


Fig 18 Artificial ears which simulate the acoustical impedance of the average human ear (From Rappaport M B and Sprague H B)[13]

design, with a length of 9 in for each ear and a bore diameter of 3/16 in An arbitrary tubing length, J, of 26 in was selected, and frequency response curves were obtained Essentially, the length of the tubing may be discounted in this experiment because a comparison is made between tubes of equal length but different caliber

Figure 19 shows the response curves of tubing whose caliber varies between 1/4 and 3/64 in The abscissa of the graph is expressed in frequency or cycles per second of the applied tone, and the ordinate is expressed both in dynes per square centimeter and in the equivalent number of decibels above the threshold reference of 0 0002 dynes per square centimeter

The family of curves shows

1 Tubing with a 1/8 in bore is the most efficient from 115 down to 40 cycles per second

2 Tubing with a 3/64 in bore is most efficient from 20 to 40 cycles per second but the efficiency drops rapidly above 40 cycles per second

3 Mitral diastolic murmurs the first, second, and third heart sounds some systolic murmurs and the atrial sound generally fall in the low frequency portion of the spectrum (20 to 115 cycles per second) The usual commercial stethoscope tubing which has a 3/16 in bore is less efficient in the range between 20 and 115 cycles per second by an *appreciable number of dynes per square centimeter of sound pressure which is applied* to the observer s ears

4 Resonant peak displacement occurs for tubing of equal lengths but different caliber The smaller the caliber the lower is the frequency of the first resonant peak the same pattern persists thereafter

5 Between 200 and 750 cycles per second the difference in efficiency is not as great although the 1/8 in tubing seems to give a slightly better result This portion of the auscultatory spectrum includes the high pitched and medium pitched murmurs

6 Above 750 cycles per second the tubing with the 1/4 in bore becomes most efficient

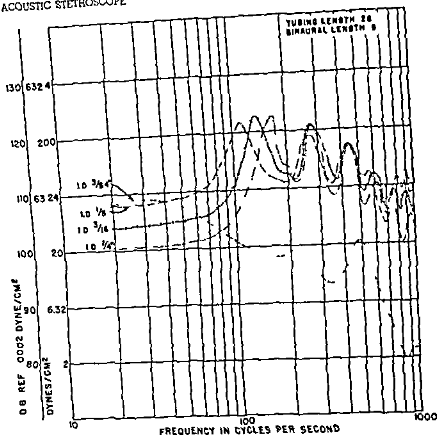


Fig 19 Re po se curves of a aco stic stethoscope in wh ch the only variable is the cali be of the t bi g I D = inter al d meter (From Rappaport M B and Sprague H B)[13]

and the $^3/_{64}$ and s in tubings are least efficient This portion of the frequency spectrum has negligible utility in cardiac auscultation rales do have some frequency components in this portion of the spectrum

A $^1/_8$ in bore in the metal binaurals instead of the usual $^3/_{16}$ in would further increase stethoscope efficiency for cardiovascular auscultation

EFFECTS OF IMPROPER FITTING OF STETHOSCOPE TO EARS ON AUSCULTATORY EFFICIENCY

In 1952 two of us (M B R and H B S)[16] investigated the effects of leaks at the observer s ears when improperly fitted ear pieces were employed on the binaural attachment of acoustic stethoscopes The instrumentation for evaluating the effects of tubing bore shown in figures 17 and 18, was used in this experiment also

It has been shown that the optimal acoustic stethoscope should have a binaural with a $^1/_8$ in caliber instead of the usual $^3/_{16}$ in which is common to most commercially available stethoscopes The rubber tubing which is interposed between the chest piece and the binaural should also have a cal iber of $^1/_8$ inch Furthermore, the tubing should be as short as possible, con

sistent with convenient handling Our experience indicates that rubber tubes approximately 10 inches long allow adequate maneuverability for most clinical applications Therefore, a stethoscope with 10 inch rubber tubes was selected to evaluate the effects of leaks at the ears

Figure 20 shows the frequency response curves for various types of leaks at the binaural ear pieces of an acoustic stethoscope Curve A occurred when

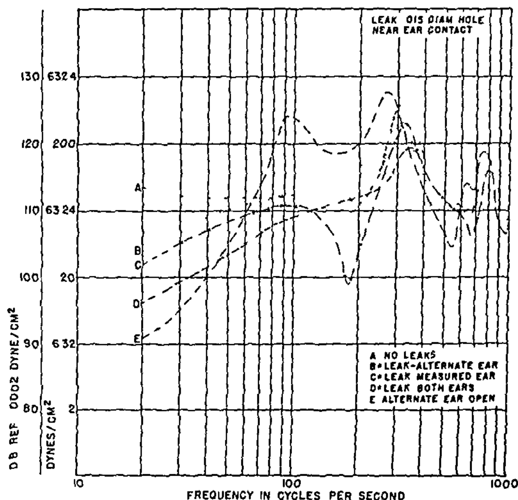


Fig 20 Frequency response curves of an acoustic stethoscope for various types of leaks at the binaural earpieces of an acoustic stethoscope (From Rappaport M B and Sprague H B) 10

no leaks were present at the ears Curve B resulted when a leak was made at the artificial ear which did not have the sound pressure meter connected to it The magnitude of the leak was a hole 0 015 in in diameter, as compared to a hair diameter of approximately *0 003 in It should be kept in mind* that the alternate artificial ear is acoustically connected to the artificial ear which has the sound pressure meter connected to it by way of the *stethoscope In curve C, the alternate ear was well sealed* but a 0 015 in leak was present at the artificial ear being measured Curve D resulted when a 0 015 in diameter leak was present at both ears Curve E occurred when

the alternate ear was disconnected from the artificial ear The abscissa of the graph is expressed in frequency or cycles per second over the auscultatory spectrum The ordinate is expressed in dynes per square centimeter of pressure measured at the artificial ear and the equivalent number of decibels above the average human threshold of hearing which is 0 0002 dynes per square centimeter

In the very low frequency portion of the auscultatory spectrum—below 70 cycles per second—the family of curves in figure 20 shows that

1 The maximum stethoscopic efficiency re ults when no leaks are present

2 The over all efficiency of the stethoscope is slightly higher when the leak is in the alternate artificial ear than when it is at the measured artificial ear This phenomenon is of no clinical value but of academic interest only

3 Leaks of equal size at both ears reduce the efficiency by a considerable amount and this efficiency diminishes rapidly as the frequency decreases

4 The most efficient condition in the low frequency spectrum results when one of the stetho copic ear pieces is disconnected from the ear which effectively produces a very large leak

Other important conclusions which can be drawn from figure 20 are

1 The resonant peaks for the various conditions of leak are altered throughout the auscultatory pectrum

2 The large leak which occurs when one ear piece is left open to the air improves the pparent efficiency from about 70 to 350 cycles per econd This effect is nullified by the introduction of excessive room noise which tend to mask the auscultatory sounds Also the ability to hear auscultatory sounds with one ear is less than it is with two ears

3 Small variance in tethoscopic efficiency occurs between 350 and 1 000 cycles per econd for all conditions according to measurement However a masking effect due to the increased superimposition of room noise on the auscultatory sounds when leaks are present is most severe in this portion of the auscultatory spectrum

4 It is imperative to have well fitted stethoscopic ear pieces for optimal auscultatory efficiency as all types of cardiovascular sound and murmurs are heard less clearly when leaks are present

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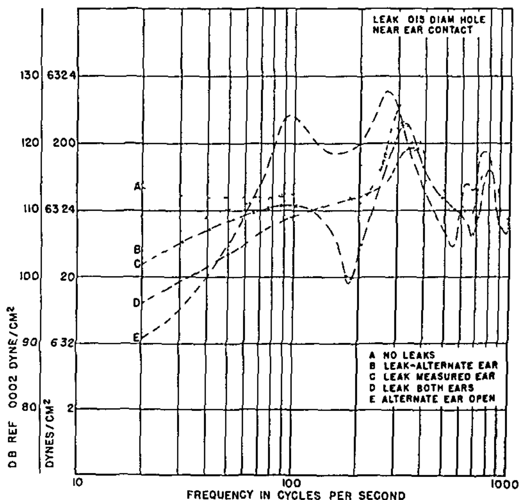


Fig 20 Frequency response curves of an acoustic stethoscope for various types of leaks at the binaural earpieces of an acoustic stethoscope (From Rappaport M B and Sprague H B)[16]

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6 / Combined Amplifying Stethoscope Phonocardiograph Electrocardiograph and Sphygmograph

INTRODUCTION

From the material presented thus far, in which the factors involved in auscultation are considered from a physiologic and physical point of view, we may safely conclude that the human auditory system, when operating in conjunction with an acoustic stethoscope, has many faults We have suggested several modifications in design for acoustic stethoscopes, with the purpose of increasing the over all auditory sensation efficiency for improved auscultation especially for threshold levels[2] No doubt exists that in the *future additional* minor improvements may be made However, after a thorough study of its physical behavior, it appears that there is little room for radical improvement because of the inflexible properties common to the instrument as well as to human hearing

Such a prediction concerning the future of the acoustic stethoscope appears bold but if average hearing is an unalterable constant, there remains only the acoustic stethoscope as the adjustable compensator for the limitations of normal hearing in auscultation The physical make up of an acoustic stethoscope is not very flexible this is true of most pure acoustic systems Until recently a somewhat similar situation existed in the science of acoustics Little progress could be made in the study of acoustics, or in the psychology and physiology of *hearing* beyond a certain point, because of the inflexibility of the purely acoustic or acoustic mechanical instruments which were available This situation becomes immediately apparent if the text books by Fletcher[3] Stevens and Davis[4] and Wever[5] are studied With the adaptation of electronics to *acoustics* and allied studies in the psychology and physiology of hearing a new approach to the subject has been opened

In a similar manner the application of electronic, electromagnetic, piezo electric and capacitive devices to auscultation has made possible a practically unlimited degree of freedom in the control of the over all performance of pick up device or transducer *and ear*, whereas in the case of the purely acoustic stethoscope flexibility in the control of this performance is limited In other words there is sufficient freedom of adjustment in these electronic devices to allow certain modifications to be made in order to compensate for some of the auscultatory failings of the human auditory system

auscultation, and their clinical application The acoustic stethoscope and the electrical amplifying stethoscope and stethograph, Am Heart J, 21 257, 1941
10 Barss W R Lade W F and Fitzgerald E B Stethoscopes Boston M & S J, 195 116 1926
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13 Rappaport, M B, and Sprague H B The effects of tubing bore on stethoscope efficiency, Am Heart J 42 605, 1951
14 Beranek L L Acoustic measurements New York John Wiley & Sons Inc 1949
15 Olson H F Elements of acoustical engineering New York D Van Nostrand Co Inc 1947
16 Rappaport M B and Sprague H B The effects of improper fitting of stethoscope to ears on auscultatory efficiency Am Heart J 43 713 1952

6 / Combined Amplifying Stethoscope Phonocardiograph Electrocardiograph and Sphygmograph

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duced electronic types of amplifying stethoscopes All of these devices altered the quality and character of the sounds so much that auscultatory technic had to be relearned and the over all improvement of the amplifying stethoscope as a clinical instrument was nullified In 1941, two of us (M B R and H B S)[10] described the first amplifying stethoscope which did not alter the quality and character of the perceived cardiovascular sounds

Many investigators, realizing the clinical limitations of human hearing made early attempts at recording the cardiovascular sounds [11 43] This study has been continued by many others [41 1 5]

Hurthle,[11] in 1893, was the first to register the heart sounds graphically He connected a microphone to a nerve muscle preparation, via an induction coil this enabled him to register on a smoked drum the apex cardiogram and the first heart sound In 1894, Einthoven and Geluk [13] substituted a capillary electrometer for the nerve muscle preparation These early experiments were primitive and actually antedated electrocardiography

The earliest recording stethoscope that had some clinical value was described by Einthoven in 1907 [15] (Fig 21) A typical stethoscope chest piece

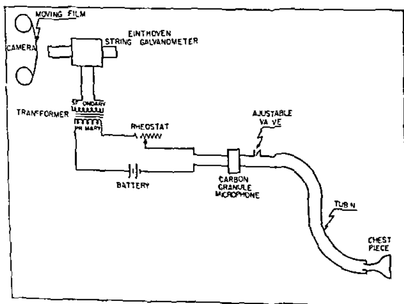


Fig 21 Sch mat c sk tch of E tho en s phonoca d og aph (From Rapp port M B and Sp ag H B) 10

was employed for accumulating the sounds, which were directed to a carbon granule microphone Interposed between the chest piece and the microphone was an adjustable leak valve which regulated the amount of low frequency heart sound reaching the microphone The microphone performed the function of converting the minute sound pressure variations in the tubing into electrical pulsations, the intensity of which was adjustable by means of the

In spite of the fact that such unlimited compensatory adjustments may make certain decided improvements in the form of increased audibility of some physiologic and pathologic cardiovascular sounds, some of the inherent shortcomings of human hearing cannot be overcome This auscultatory problem is summed up by Wiggers [6] as follows

> Most of the vibrations of the chest wall inaugurated by the two sounds of the heart have an amplitude and frequency which approach the auditory limit, indeed it is probable that some of the vibrations are incapable of being recognized by the ear While the auditory appreciation for slow vibrations of low frequency lasting only for brief moments can be cultivated by practice and training the average ear and brain have some difficulty in differentiating the finer differences in time frequency and form shown to exist in graphic records When waves of several frequencies intermingle the ear according to the inclination or training tends to pick out one group and to suppress the other

In addition to what we may consider the normal faults of the human auditory system in auscultation, another important condition must be taken into consideration Often a physician who is under the impression that his hearing is normal discovers that an audiogram shows peaks and valleys in the auscultatory frequency spectrum Many times these valleys fall well below the threshold of hearing for some of the important sounds When an average of a large number of subjects is taken, a cancelling out of these peaks and valleys occurs, and as a result the average audiogram is more smooth or uniform than that of many individuals

Such shortcomings of hearing may be surmounted by supplementing auscultation with graphic recording A graphic recording system designed to register the required frequency spectrum is immune to the many auditory defects of the ear

HISTORY

At the turn of the century, attempts were made to improve auscultation by means of acoustic and electro acoustic devices The experiments as a whole ended in failure when purely acoustic means were employed An electro acoustic system was devised which did succeed in amplifying the sounds, but excessive distortion made the instrument useless The electro acoustic stethoscope operated on a principle similar to that of the telephone it consisted of a carbon granule microphone which modulated an electric current, and this, in turn, excited a telephone receiver

Successful amplification of the cardiovascular sounds was made possible by the development of the audion tube Although the history of electronic amplification dates back to the practical results obtained by de Forest [7] in 1907, the audion tube amplifier was not reliable for clinical auscultatory application until much later

One of the earliest, practical amplifying stethoscopes was developed by the Western Electric Company [8, 9] in 1924 Several other experimenters pro

Williams apparatus Trendelenburg's[40 43] phonocardiograph employed a condenser type of microphone, and Sell's[44 4] apparatus employed an electrodynamic type, operating with a multistage amplifier and a moving coil mirror galvanometer However, even with the improvements incorporated by Williams, Sell, Trendelenburg Gamble and Replogle, and others, the phonocardiograph did not reproduce cardiovascular sounds with the fidelity with which they could be heard with the ordinary acoustic stethoscope or by immediate auscultation The relationship between hearing and recording was extremely vague

In our work[53 4] at first, we used a piezoelectric crystal microphone with a multistage amplifier and a moving coil mirror galvanometer, later substituting an electrodynamic microphone * By this method the cardiovascular sounds could be reproduced without the slightest noticeable alteration in quality, using the apparatus as an amplifying stethoscope, and, in addition, the sounds could be recorded simultaneously with the electrocardiogram and the sphygmogram

GENERAL CONSIDERATIONS

An amplifying stethoscope, as the name indicates, is an apparatus which is capable of increasing the intensity of sounds, this may be accomplished either electrically piezoelectrically electronically acoustically, mechanically, electromagnetically, or by any combination of these methods The following are some of the most important factors that should be taken into consideration in the design of an amplifying stethoscope

1 The apparatus must be capable of reproducing the sounds like an acoustical stethoscope It mu t not alter the qual ty and character of the ounds from what is normally heard with an acoustical stethoscope of accepted performance

2 The amplifying stethoscope must be free of any inherent noise and be immune to the influence of external electrical wiring diathermy machines x ray apparatus or household appliances Inherent noise in an amplifying stethoscope raises the threshold of hearing

3 Normal room noises and vibrations should not affect the operation of the apparatus If the amplifying stethoscope is en itive to such extraneous ounds the thre hold of hearing is rai ed

4 It should be po ible to control the loudness of the sounds by means of a calibrated loudne s control which is ea ily adjustable

5 The apparatus must be free of feed back howls and squeals when the chest piece or ound pickup u it is brought into the vicini y of the hearing device or raised away from the precordium

6 The apparatus must be capable of withstanding rough handling

7 It may be advantageous to make the amplifying stethoscope independent of house current but battery replacement is a nui ance

8 The frequency respon e characteristic of the amplifying stethoscope should over

* The in trumentation used was made available by the Sanborn Company Waltham Massachusetts.

rheostat, these electrical pulsations were allowed to pass through the primary winding of the transformer The transformed electrical pulsations were led off the secondary winding of the transformer and passed through the metallic coated quartz string of the galvanometer The purpose of the transformer was to prevent the direct polarizing current, produced by the battery, from passing through the galvanometer string The tension of the galvanometer string was greater than that employed in making electrocardiograms, this allowed the string to sense the higher pitched cardiovascular sounds The oscillations of the string were photographed in the usual electrocardiographic manner, thereby producing a phonocardiogram, as Einthoven preferred to call the graphic sound record Einthoven could listen to the sounds by placing a telephone receiver across the secondary winding of the transformer

Although the Einthoven phonocardiograph was capable of recording the cardiovascular sounds graphically, it was far from being a satisfactory clinical apparatus for the following reasons

1 Einthoven could not produce a smooth base line when no physiologic sounds were present because extraneous noises entered the system through the leak valve The carbon particles in the microphone also produced extraneous noises as is characteristic of such microphones

2 The transformer employed by Einthoven introduced considerable distortion understanding of audio transformer performance and design was practically unknown at that time

3 The cardiovascular sounds heard by placing a telephone receiver across the secondary winding of the transformer were so distorted as to be hardly recognizable

Einthoven, although decidedly handicapped by the lack of suitable apparatus, did succeed in obtaining much valuable information concerning the nature of cardiovascular sounds

The earlier investigators met with some success in registering cardiovascular sounds by the direct method, which is a system employing the physical principles of the human eardrum The apparatus is usually composed of a stethoscope bell connected to a sensitive membrane by means of a rubber tube The movements of the membrane produced by the cardiovascular sounds are recorded by means of an optical system focused on moving photographic film The membrane in the direct system of heart sound recording may be compared with the tympanic membrane of the ear, and it is usually constructed of either thin rubber, mesentery from the guinea pig, gelatin, soap film, or isinglass

One of the most successful early phonocardiographs was devised by H B Williams [43] It consisted of an electromagnetic telephone receiver operating in reverse, as a microphone The minute electrical pulsations were then intensified by means of a four stage audion amplifier and recorded with an Einthoven string galvanometer The Western Electric Stethophone described by Gamble and Replogle [1 9] operated on essentially the same principle as the

electrical pulsations These minute electrical pulsations are passed *into* an *electron tube* amplifier, and the amount of amplification is regulated by a *loudness* control The audiophones, which are connected to the output of the amplifier, reconvert the strengthened electrical pulsations into sound energy

PHONOCARDIOGRAM

A moving-coil (d Arsonval) galvanometer which is capable of responding to the frequency spectrum of the sounds encountered in auscultatory work is also connected to the output of the sound amplifier The galvanometer coil which carries a mirror, deflects in response to the intensified electrical pulsations A beam of light is reflected from the mirror onto *uniformly* moving film or bromide coated paper in a kymograph The movement of the mirror correspondingly moves the light beam across the sensitized film or paper The combination of the longitudinal motion of the film and the transverse movement of the light beam produces a graph of the sounds, plotted against time (a phonocardiogram)

ELECTROCARDIOGRAM

The electrocardiogram is obtained by directing the action potentials of the heart into a suitable leadswitching and calibrating circuit, amplifying or strengthening the minute currents and feeding them into another moving coil galvanometer, the frequency response characteristics of which are suited to electrocardiography By means of a suitable optical system, the movements of the galvanometer coil are recorded on the same sensitized film or paper on which the phonocardiogram is registered, thereby producing a simultaneous electrocardiogram and phonocardiogram The galvanometer and optical components must be so arranged that parallax does not exist Otherwise simultaneous cardiovascular functions register with a temporal displacement toward each other Vertical time marking lines, together with horizontal amplitude lines are superimposed optically over the electrocardiogram section Other arrangements of a more complex optical nature are employed where the electrocardiographic recording is by beam instead of shadow and the time and amplitude lines are then registered as black lines on a white background The latter form of registration is more desirable, as the beams can cross each other on the sensitized film and more than two recording channels may be used without excessively widening the sensitized paper or film

MICROPHONE

The invention of the telephone in 1876 marked the beginning *of the development* of devices capable of converting sound energy into electrical energy The fundamental principles of nearly all microphones (technically known as electro acoustic transducers) which are in use today *date back to* Bell s time but until recently, these microphones could not be used because

come as much as possible the normal or pathologic hearing defects which may reduce acuity in auscultation

The phonocardiograph, or heart sound recording mechanism, must be capable of performing the following functions

1 It must record graphically all of the sounds that are audible with the acoustic stetho scope

2 It must be capable of registering sounds that are easily missed as a result of masking and other hearing characteristics that limit optimal auscultation

3 When there are no sounds such as during normal systole and diastole the base line must be reasonably smooth

4 The phonocardiograph must be immune to all types of external electrical interference

5 The instrument must be capable of simultaneously registering at least one other cardiovascular event such as the electrocardiogram or sphygmogram for timing the events in the phonocardiogram When certain pathologic conditions are present the phono cardiogram may be meaningless or confusing without a simultaneously registered electro cardiogram or sphygmogram

6 The electrocardiogram and sphygmogram must conform with accepted standards and requirements

GENERAL COMPONENT RELATIONSHIPS (FIG 22)

Amplifying stethoscope

The amplifying stethoscope consists of a microphone which serves to con vert the sound energy picked up at the precordium into equivalent, minute,

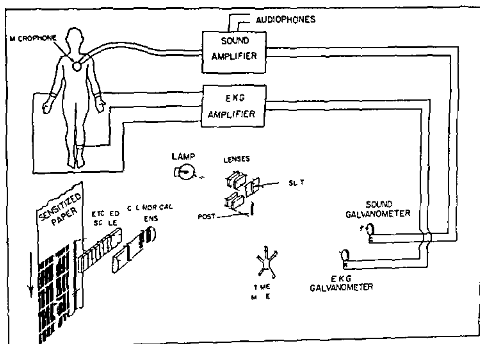


Fig 22 Schematic diagram illustrating the inter relationship of the various components of an apparatus capable of producing a simultaneous electrocardiogram and phonocardiogram and of amplifying the loudness of the cardiovascular sounds (From Rappaport M B and Sprague H B) [10]

funnel surrounded the microphone button The microphone was operated by suspending the unit from a cushioned pulley and fastening the suction bell to the patient's chest This arrangement placed the microphone button in contact with the chest and thereby, provided for transmission of the sound vibrations to the coil The vibrating coil, in turn, cut the lines of force produced by the fixed magnet, setting up an electromotive force in the coil The coil was electrically connected to a transformer, followed by an amplifier as in the Williams apparatus

The major improvement in the Sell microphone was in the construction of the chest piece, which eliminated to a large extent the extraneous noises caused by slight shifting of the button on the chest The other undesirable features of the Williams microphone were also present in the Sell microphone for the general nature of the two was the same

The suction chest piece used by Sell functioned well when there was no heavy growth of hair on the chest However, when hair was present, the suction could not be maintained, and the chest had to be shaved The application of an oily substance such as vaseline, to the patient's chest often helped to maintain the vacuum if the hair was not too dense

Trendelenburg[1, 2] used a microphone which operated on the *electrostatic* principle It is better known as a condenser microphone *In its simplest* form a condenser microphone comprises a light, stretched, metallic diaphragm spaced at a short distance from another parallel, metallic plate which acts as a fixed electrode A steady, direct-current, polarizing potential, such as that used with the carbon granule microphone but of considerable higher value (usually about 135 volts) is placed across the parallel plates through a series resistance The minute, alternating variations of capacity caused by the movements of the diaphragm which, in turn are caused by the heart sounds create a variable impedance which produces a pulsating current that is equivalent in wave form to that of the heart sounds The generated pulsating currents of electricity flowing through the resistor which is connected in series with the microphone set up an equivalent pulsating voltage across the resistor and this is fed to an electronic amplifier and intensified

The Trendelenburg microphone employed a suction chest piece similar to that used by Sell with the exception that sound vibrations of the chest wall were transmitted to the diaphragm through a column of air instead of by direct mechanical contact Trendelenburg's microphone was far superior to the previously mentioned types in that it eliminated the electromagnetic feed back howl when an electromagnetic ear piece was employed However two major faults still remained namely

1 The characteristically low sensitivity of a condenser microphone of this type necessitates the use of an electronic amplifier producing considerably more amplification and a higher degree of instability and inherent noise

2 When relatively high pressures are set up in the air column of a condenser micro

they were not sufficiently sensitive The perfection of the vacuum tube amplifier in recent years has made it possible to take advantage of many of the desirable properties of these electro acoustic transducers

A microphone is an electro acoustic transducer which, when actuated by the power in an acoustic system, will deliver power to an electrical system, and the wave form in the electrical system will correspond to that in the acoustic system

When Einthoven performed his experiments with the phonocardiograph, electronic amplifiers were not known As a result, he was forced to employ a microphone which was not dependent on electronic amplification The best transducer available was the carbon granule microphone[130, 131] As previously noted, this type of transducer, when employed for phonocardiographic purposes, possessed many limitations H B Williams improved the Einthoven system by substituting a magnetic type of telephone receiver to the diaphragm of which a nonmetallic button was cemented The button was placed in contact with the patient's precordium, so that the sound vibrations were transmitted mechanically to the diaphragm The vibrating diaphragm, in turn, induced an equivalent electromotive force in the coil of the electromagnet of the telephone receiver The coil was electrically connected to a step up transformer which fed into an electronic amplifier

Although Williams' microphone was a decided improvement over the one employed by Einthoven in that it eliminated the background noise caused by the carbon granules, it possessed some undesirable qualities

1 The Williams microphone required a coupling transformer between the microphone and amplifier If a transformer is followed by a highly sensitive amplifier minute alternating current interference may be picked up from electrical appliances by the transformer amplified and superimposed on the heart sounds This difficulty which was characteristic of the Williams phonocardiograph can be circumvented at the present time by improved transformer technics and electromagnetic shielding (see p 74)

2 For auscultation a telephone receiver was coupled to the binaural attachment of a stethoscope the receiver was connected to the output stage of the electronic amplifier When the telephone receiver was not kept at a considerable distance from the microphone an electromagnetic feed back took place and a howling sound was heard Improved shielding technics eliminate this fault

3 Considerable difficulty was encountered in subduing extraneous noises caused by slight shifting of the microphone contact button on the patient's precordium unless the microphone was carefully fastened with adhesive tape This is an awkward clinical procedure

4 The auscultatory sounds heard through the binaurals did not have the quality and character of those heard with an acoustic stethoscope The reasons for this will be apparent from theory and data which will be discussed later (pp 67 68)

Sell,[44, 4] in an endeavor to improve upon the Williams apparatus, constructed an electromagnetic microphone It consisted of a magnet and a movable coil The coil was physically connected to a button which was in contact with the patient's precordium A double walled, suction type of

4 The impedance of a crystal element is such that a coupling transformer is not required

5 The output level (voltage output from the microphone versus sound pressure excitation) is high as compared with that of other types of microphone elements

In 1940, Stordal[234] described the phonocardiographic apparatus which was used by Mannheimer This apparatus employed a piezoelectric microphone of a rather unique construction * It was composed of a chamber, one end of which was covered with a rubber diaphragm The rubber diaphragm was placed on the precordium and movements corresponding to the sound vibrations were produced in the diaphragm The movements of the rubber diaphragm in turn produced pressure variations on a directly operated piezoelectric crystal which transformed the sound pressures into electricity Mannheimer also used a closed chamber, in the form of a bell type chest piece, on the same microphone, and he stated that the phonocardiograms were generally registered without the bell To measure absolute sound amplitude by means of calibration he used the bell

The phonocardiograph which we (M B R and H B S)[10] described in 1941 † contains a piezoelectric microphone of a different type *The heart of* the microphone is a cartridge or diaphragm *piezoelectric unit* (*Fig* 23)

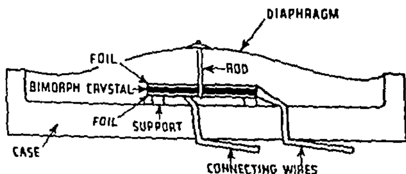


Fig 23 Cross section sketch of the piezoelectric crystal microphone cartridge (From Rappaport M B and Sprague H B) 10

It consists of a curvilinear *diaphragm* which is coupled directly to a bimorph rochelle salt crystal[13, 130] *through* a tiny connecting rod, the crystal and connecting rod are located *in the interior* of the cartridge When an actuating force in the form of *sound pressure* is applied to the diaphragm, proportional stresses are set up in the crystal, and it *transforms these stresses* into equivalent electrical potentials which are *conducted* away from the crystal by means of metallic foil electrodes in contact with it The natural frequency of the bimorph crystal is *approximately 10 000* cycles per second When a diaphragm is coupled to the crystal (*Fig* 23) *the* natural frequency of the

* Manufactured by Sure *Brothers* Chicago Illinois U.S.A (model number 66)

† Manufactured by the Sanborn *Company* Waltham Massachusetts U S A (the Stetho Cardiette)

phone of the Trendelenburg type, as for example when the open bell chest piece is applied to the patient s chest, the diaphragm tends to be forced over to the other plate electrode Because there is a polarizing potential between the two electrodes of the micro phone pitting of the metal takes place at the point of contact every time the electrodes touch After a number of such contacts, the microphone may become inoperative

The electrical impedance of a microphone may vary anywhere between a few ohms and many hundreds of thousands of ohms, depending on the type For satisfactory operation, the electrical impedance of a microphone must match the impedance of the following circuit element In the electromagnetic microphone, such as that of Sell or Williams, the impedance of the coil must be properly matched The impedance of such a coil (depending on the de sign) may vary between a few ohms and several thousand Therefore, in order to match such an element to an electronic amplifier and to retain maximum efficiency and a minimum of distortion, a coupling transformer must be introduced in such a way that the primary winding of the trans former matches the microphone coil and the secondary winding of the transformer matches the amplifier

In a condenser microphone, such as the Trendelenburg type, the inherent impedance is extremely high, and of the same order as the input of an elec tronic amplifier As a result, no coupling transformer is necessary Thus, some of the previously mentioned difficulties which were encountered by Sell and Williams when a transformer was placed in so delicate a location were eliminated by Trendelenburg

In the early 1930s, the electronic industry started to perfect the piezo electric or crystal microphone,[13, 133] for radio and public address systems A piezoelectric microphone is one in which the electrical output is dependent on the stresses set up in a crystal of rochelle salt, quartz, or tourmaline In such a device the output potentials are proportional to the stresses which the sound pressures induce Rochelle salt, which is sodium potassium tartrate with four molecules of water of crystallization, exhibits the largest piezo electric activity of all known crystals, more than one thousand times that of quartz and tourmaline

Two general classifications of piezoelectric microphones are commonly used in electronics, namely, the directly actuated and diaphragm actuated types In a directly actuated piezoelectric microphone, the sound pressure impinges directly upon the crystal, and in the diaphragm actuated type, the sound pressure actuates a diaphragm which is mechanically coupled to the piezoelectric crystal

The piezoelectric crystal element possesses many characteristics which make it suitable for phonocardiography

1 The piezoelectric crystal element is inherently free of noises

2 The crystal element is small is light in weight and requires only simple electro static shielding

3 It does not require any polarizing potentials

2 The high pass acoustical filter protects the crystal from sudden high pressures of extremely low frequency such as those which are produced by applying the microphone to the chest

In the logarithmic microphone, the acoustic high pass filter, filtering characteristics of which are different with respect to frequency from those of the acoustic filter in the stethoscopic microphone, introduces, in addition the so-called 'logarithmic frequency response"

A filter, whether it be electrical, mechanical or acoustic, is a system which will freely pass desired frequency spectrums, and highly attenuate neighboring undesired frequency spectrums Campbell[137] in 1922, is credited with having developed the electrical filter, Stewart,[138] also in 1922, was the first to describe the acoustic types The mechanical filter has been employed in various kinds of apparatus for many centuries The electrical wave filter action has been further investigated by Zobel,[139] Johnson,[140] and Shea,[141] the acoustic filter by Mason,[14] and the mechanical types, by Crandall[142] The most complete investigations on filtering action have been carried out for the electrical type However, analysis of the acoustic and mechanical filters shows them to be similar to those of the electrical type

Figure 25 shows three equivalent filters which possess low pass charac

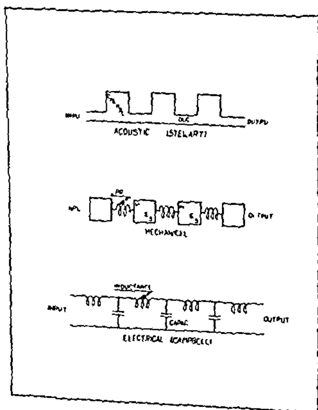


Fig 25 Equivalent low pass Filters (From Rappaport M B and Sprague H B) 10

combination is lowered to a few thousand cycles per second which is well above the upper frequency components encountered in auscultatory sounds

The cartridge is fitted into a case designed for phonocardiography (Fig 24A, B, and C) The microphone case proper is constructed to incorporate what is technically known as an acoustic high pass filter and two types of filters are used to produce what we [10, 4] have termed *stethoscopic* and *logarithmic* responses (see p 103) The acoustic high pass filter in the stethoscopic microphone serves two essential purposes, namely

1 It eliminates the effects on the microphone of the extremely low frequency vibrations or motions of the chest wall which are not wanted These low frequency vibrations may be studied with the *linear microphone* [10, 54] (see p 105)

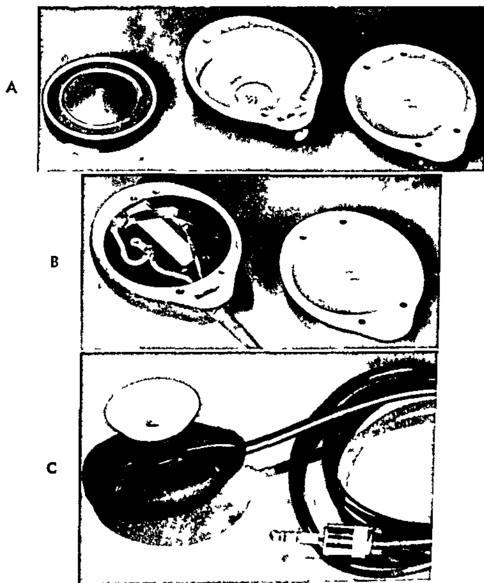


Fig 24 A Microphone case with built in acoustic high pass filter and piezo electric cartridge B Manner in which cartridge is inserted into microphone case C Completely assembled microphone with a large open bell chest piece (From Rappaport M B and Sprague H B) [10]

ternal air and the air column which was under heart sound excitation caused severe extraneous noise

In the piezoelectric microphone we used,[10] the acoustic filter channels are so constructed that they connect with a chamber hermetically sealed into the microphone case, the chamber acts as a substitute for the external air. This type of acoustic filter has made possible the necessary degree of high pass filtering action on the heart sounds and chest wall vibrations, without introducing the slightest traceable extraneous noise

The four bells, or chest pieces, in figure 10 are used in conjunction with the microphone. Each bell, as previously described, produces a different additional degree of filtering or selective attenuation of certain frequencies. The desired bell may be screwed into the microphone case

In spite of the fact that the piezoelectric microphone, with its rochelle salt crystal has many excellent characteristics when used in phonocardiography and auscultation, a few undesirable characteristics are inherent. Humidity has severe effects on the rochelle salt. If the microphone is used in climates where the humidity is low (less than 30 per cent) the crystal gradually dehydrates and becomes a powder. In localities where the humidity is generally high (more than 84 per cent) the crystal gradually dissolves. Manufacturers of piezoelectric crystal elements apply protective coatings to the crystals but experience has shown that these coatings do not afford permanent and complete protection and the life of the microphone is limited

Piezoelectric crystal microphones are affected by temperature also. The sensitivity of a crystal varies to an appreciable degree with room temperature and as a result permanent intensity calibrations cannot be attained. If the microphone is subjected to temperatures higher than 115° F the crystal is permanently damaged

A crystal microphone cannot withstand rough handling. If dropped, it is likely to be permanently damaged. The dropping of a microphone in clinical phonocardiography and auscultation is not an uncommon occurrence

A recent addition to the family of piezoelectric substances is a ceramic material made from barium titanate.[144,146] It is rendered piezoelectric in the manufacturing process by being permanently polarized with 40,000 to 60 000 volts per centimeter for a period of from several minutes to an hour. Accurate data on the physical constants of barium titanate transducers is not available. However it is known that microphones which use barium titanate are consistently less sensitive by approximately 18 decibels than those using rochelle salt, when both have equal capacitance and mechanical compliance. An 18 decibel loss in sensitivity must be compensated for by increasing the electronic amplification 18 decibels or eightfold. this amount of additional amplification brings in objectionable amplifier noises which are most annoying when the instrument is used as an amplifying stethoscope. Also the barium titanate is just about as fragile as the rochelle salt crystal

teristics These filters are known as two element types because each element contains two components The condenser in the electrical circuit, the chamber in the acoustic filter, and the spring in the mechanical filter perform corresponding functions The inductance in the electrical filter performs a function similar to that of the duct in the acoustic filter and the mass in the mechanical filter Figure 26 shows three equivalent filters which have high

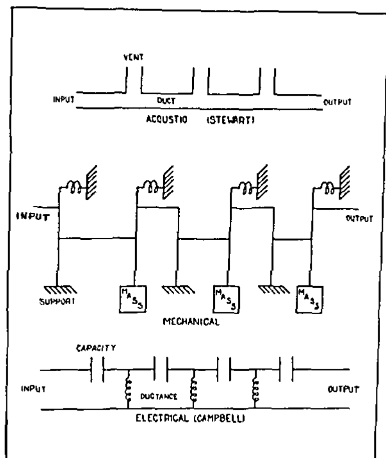


Fig 26 Equivalent high pass filters (From Rappaport M B and Sprague H B)[10]

pass characteristics Theoretically, a low pass filter should allow the passage of electrical currents, velocities, and volume currents, in electrical, mechanical, and acoustic systems, of all frequencies lying between zero and the filter cut off frequency, which is dependent on the constants of the filter On the other hand, a high pass filter allows the passage of currents, velocities, and volume currents, in electrical, mechanical, and acoustic systems, of all frequencies above the filter cut off frequency

The carbon granule microphone employed by Einthoven in his phonocardiograph possessed a simple, but crude, sort of high pass acoustic filter Einthoven accomplished the filtering process by introducing an adjustable valve (Fig 21) The fact that Einthoven allowed access between the ex

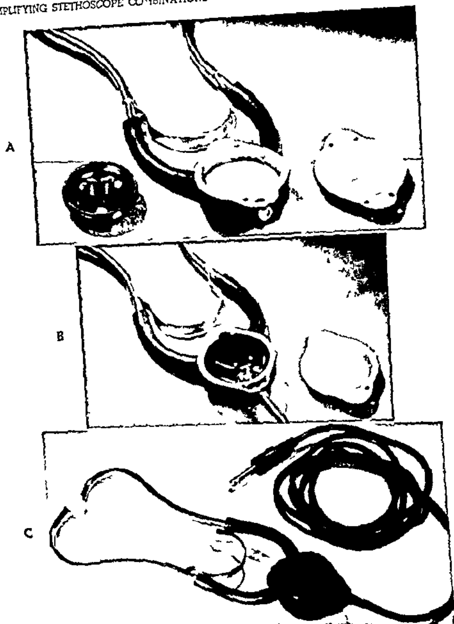


Fig 27 A Audophone with cover and electromagnetic phone removed B Manner in which electromagnetic phone is inserted into audophone C Assembled audophone (From Rappaport M B and Sprague H B)[10]

to an acoustic system, and the wave form of the sound pressure in the acoustic system corresponds to the wave form in the electrical system Telephone receivers may be divided into four general classes (1) electromagnetic (2) electrodynamic (3) electrostatic and (4) piezoelectric Complete descriptions of the electromechanics of these electro acoustic transducers may be found in most textbooks on applied acoustics,[13 130 147] telephony,[130]

On the other hand, barium titanate has excellent temperature sensitivity stability, and can withstand high temperatures

In order to produce an amplifying stethoscope and phonocardiograph which can be permanently calibrated, is rugged, and possesses the many desirable characteristics of a rochelle salt piezoelectric microphone, one of us (M B R) designed a modified form of the dynamic microphone * The sensing element in the microphone is a diaphragm to which is cemented a light coil of wire which is in a strong magnetic field This type of transducer requires a step up transformer like the Williams and Sell microphones and is equally susceptible to interference such as was described However, Rappaport was able to shield the transducer element and transformer with a recently developed iron alloy known as mu metal which is specially heat treated This form of shielding overcame the difficulties which plagued Williams and Sell Modern transformer technics in core construction and coil winding also have contributed toward reducing the interference

The sensitivity of the combined dynamic microphone and transformer is about 6 decibels or twofold less than that of the rochelle salt piezoelectric microphone, a twofold increase in electronic amplification is not enough to bring in the objectionable background disturbances in the amplifier The microphone is rugged and can withstand considerable mishandling The sensitivity does not vary with temperature, can withstand high temperatures, is not affected by high or low humidity, and does not require a high pass filter to protect the sensing element Logarithmic and stethoscopic responses are attained by electrical means as will be explained later (see p 102) The microphone case and chest pieces are similar to those used in the piezo electric microphone except that the cavity volume is less in the dynamic microphone Cavity volume can alter the frequency response of the heart sound microphone and, thus, is an important parameter in the design

Audiophone

In an amplifying stethoscope, the audiophone is the component which converts or transduces the amplified electrical pulsations into sounds such as those which are normally heard with the acoustic stethoscope (*Fig 27A, B, and C*) [10] The three fundamental components of an audiophone are the electro acoustic transducer, the case, and the binaural

The electro acoustic transducer is an electromagnetic telephone receiver of the diaphragm armature type which can produce sound levels as high as 125 decibels above the threshold level of 0 0002 dyne per square centimeter without overloading There are certain auscultatory sounds which attain such magnitudes [6] *A telephone receiver is an electro acoustic transducer* which, when actuated by an electrical system (the amplifier, in the case of the amplifying stethoscope or phonocardiograph), supplies sound pressure

* Manufactured by the Sanborn Company Waltham Massachusetts Used in the Twin Beam Cardiette and a later model of the Amplifying Stethoscope

effects which may be considered equivalent to a resistive component This effect is of small consequence in cardiovascular auscultatory work, as the frequencies which are encountered do not exceed 1 000 cycles per second On the other hand, at low frequencies, leakage which may occur in an improperly designed audiophone gives rise to a reactive and resistive component This factor may vary from person to person if the ear pieces of the binaural are not properly placed or fitted It is an important consideration in the action of the acoustic, as well as of the amplifying stethoscope An hermetically sealed audiophone also eliminates room noises which seem greatly exaggerated, the same holds true for the acoustic stethoscope

Amplifier

The phonocardiograph electrocardiograph amplifier we used (Fig 28) [10] comprises two amplifier channels of entirely independent and different electrical characteristics One channel possesses electrical characteristics which are useful for amplifying the cardiovascular sounds, whereas the other channel is suitable for amplifying the action potentials of the heart, such as those with which electrocardiography is concerned

The amplifying systems of the phonocardiograph-electrocardiograph employ thermionic vacuum tubes A thermionic vacuum tube consists of an

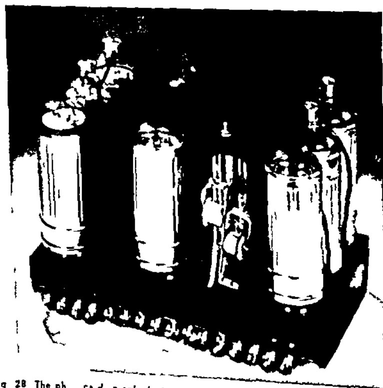


Fig 28 The ph ca d g aph l ct oca d g aph ampl fer (From Rappap t M B and Sp agu H B) 10

and electrical engineering [148] The electromagnetic type, which was invented by Bell and perfected by others, was selected by Rappaport and Sprague as the audiophone component because of its simple construction, rugged nature, ability to cover the required frequency spectrum, compactness, suitable im pedance characteristics, and its ability to reproduce the loudest auscultatory sounds without overloading and distortion

The telephone receiver first developed by Bell consisted of an electro magnet which retained a residual amount of magnetism The strength of the residual magnetic field could be modulated by the electric signal which was allowed to pass through a coil wrapped around the magnet A steel diaphragm, clamped at its circumference and placed a short distance from the pole pieces of the electromagnet, was caused to vibrate at a frequency and to a degree which was proportional to the frequency and magnitude of the electrical signal The diaphragm action, in turn, set the surrounding air into similar motion, and this produced the sound which was perceived by the ear Present day instruments operate on the same principle, but im proved materials and design have greatly increased their efficiency

The only moving part in an electromagnetic receiver of the diaphragm armature type is the diaphragm Although the system appears extremely simple from a mechanical standpoint, the theoretical analysis is complex Investigations into the theoretical performance of the telephone receiver have been made by Crandall,[143] Kennelly,[148] and Lamb [149] All agree that the mechanics of the diaphragm closely resemble those of an ideal, clamped, circular plate

In the design of the electro acoustic transducer which is employed in the audiophone, a diaphragm with a fundamental resonance peak of approxi mately 1,000 cycles per second was selected Following this resonance point, other resonances occur as the frequency is increased The fundamental reso nance, and certainly the higher frequency modes of vibration, are well above the auscultatory frequency spectrum, and, therefore, produce no ill effects

The acoustic system in an audiophone consists of the inertance produced by the binaural system and the capacitance of the volume which is excited by the diaphragm In an analysis of the action of an audiophone, the acoustic system, as well as the mechanics of the diaphragm, must be considered

In an ideal audiophone, the ratio of the sound pressure variation delivered to the ear cavity to the voltage applied to the transducer, should be inde pendent of the frequency If the assumption is made that the cavity of the ear presents a constant capacitance to the audiophone, the ratio of the pres sure built up in the ear cavity to the amplitude of the diaphragm vibrations should be independent of the frequency It is interesting that the independ ence offered by the ear cavity to the audiophone at the higher frequencies is not purely capacitive, but that it becomes more so at the lower frequencies This phenomenon is caused by the presence of standing wave systems be tween the transducer and portions of the ear cavity, as well as by absorption

well as that of the audiophone and microphone, affects the quality of the heart sounds as heard with the combination or amplifying stethoscope

The electrocardiograph amplifier channel operates on the same principle as the phonocardiograph amplifier, in that both are resistance-capacity coupled However, the electrocardiograph amplifier possesses a time constant which permits all frequencies encountered in electrocardiography to be amplified without the slightest degree of attenuation or accentuation

The amplifier system shown in figure 28 is powered by dry batteries More modern versions are shown in figures 29 and 30 which are components of the Sanborn Twin Beam Cardiette, these amplifying systems are powered from alternating current mains or house current

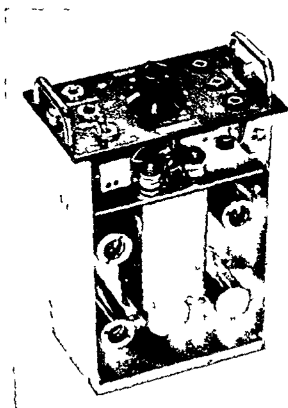


Fig 29 Photograph of phonocardiographic amplifier (Courtesy of Sanborn Company Waltham Massachusetts)

FREQUENCY RESPONSE VERSUS HEART SOUND QUALITY

A theoretical analysis of the relationship that exists between the frequency response characteristic of an acoustic or amplifying stethoscope and the quality of the sound heard by the observer is of utmost importance A

evacuated enclosure containing a number of electrodes, between two or more of which conduction of electricity through the vacuum may take place The three most essential electrodes in a thermionic vacuum tube which is suitable for amplification are the cathode, grid, and anode

The grid is interposed between the cathode and the anode, and controls the flow of current between them That is, normally, electrons from the heated cathode flow between the cathode and the anode, but if a potential is applied to the grid, making it negative with respect to the cathode, the current flowing between the cathode and the anode is reduced proportionately to the amount of grid negativity A change in grid potential has a great effect on the amount of current which flows between the cathode and the anode If a resistor is placed in the anode circuit so that the anode current variations are allowed to pass through the resistor, a proportional voltage variation takes place across the resistor Thus, the voltage variation across the plate resistor is similar to the grid voltage variation, but is amplified

When a greater degree of amplification is desired than can be obtained with a single stage or tube, audion tubes may be combined to form a multistage or cascade amplifier The tubes of succeeding stages of a cascade amplifier may be interlinked in proper sequence through some medium which transfers the intensified signal from one stage to another for additional intensification without affecting the operation of each individual stage

The anode circuit in each tube of a multistage amplifier must be closed by some form of impedance or resistance, this is also true for a single stage amplifier The signal potential that manifests itself across the impedance load is then impressed on the grid of the next tube or stage There are several types of multistage amplifiers which are classified according to the anode load impedance circuit,[150] such as resistance capacity coupled, impedance capacity coupled, direct coupled, and transformer coupled

The resistance capacity coupled circuit employed in the phonocardiograph amplifier channel[1 9] comprises the arrangement in which the anode load is a pure, high resistance The grid circuit of the succeeding stage is also composed of a pure high resistance The grid and anode resistors are linked by a condenser which acts as an isolating medium for the direct current potentials in the grid and anode circuits Should the direct current potentials which control the operation of the amplifier stages intermingle the amplifier as a whole would not function properly, if at all The signal potential, which is of an alternating character, is capable of passing through the condenser

The degree of amplification of the signal depends on the amplification provided by the tubes in the various stages, in addition to whatever attenuation may take place as a result of the relationship of signal frequency to the resistance capacity values employed for coupling The relationship is commonly known as the time constant, or frequency response characteristic of the amplifier The frequency response characteristic of the amplifier, as

chest pieces introduced different tonal qualities, as well as variations in intensity

The next step was to investigate the nature of the two most commonly used chest pieces namely, (1) the open bell, with a diameter of one inch, and (2) the Bowles, or diaphragm type, with a working diameter of one and three eighth inches

There was a slight variation in tonal quality and intensity when the most commonly used stethoscopes which employ these two chest pieces were compared The general consensus of cardiologists was that, although a noticeable difference in tonal quality existed it was not sufficient seriously to affect general auscultatory technic

We obtained frequency response characteristic curves for the various popular makes of stethoscopes which employ the two chest pieces, and a mean frequency response characteristic was calculated With this average curve as a basis, we constructed an amplifying stethoscope with a frequency response characteristic which was identical to the theoretically average stethoscope [10]

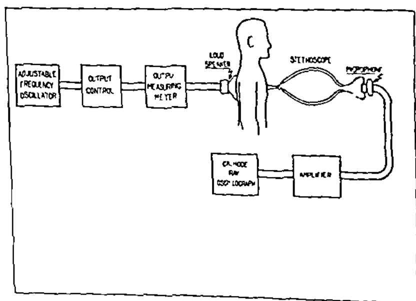


Fig 31 Sch m t c d ag m of apparat s used for omps s g frequency sponse cha act r st cs of aco st c stethoscopes and ampl fy g s thoscopes (F m Rappaport M B and Sp g e H B) 10

Figure 31 is a schematic diagram of the apparatus we employed in ascertaining the frequency response from which the mean frequency response characteristic of the acoustic stethoscope was calculated The characteristics of the loud speaker the transmissional effects of the mediums comprising the patient s chest and other such modifying factors were eliminated by the subtraction method (previously described in connection with the determina

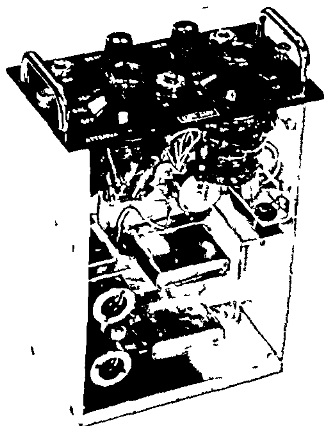


Fig 30 Photograph of an electrocardiographic amplifier (Courtesy of Sanborn Company Waltham Massachusetts)

thorough study of all available literature pertaining to amplifying, acoustic, or recording stethoscopes prior to 1941 [10] reveals a surprising lack of reference to this aspect of the subject A still more surprising fact is that there are no set standards to follow, this is certainly a unique situation when considering the importance of correct design in an instrument which is so widely used

The primary requirements for any kind of stethoscope which is to be used by a competent observer with normal hearing are as follows

1 The stethoscope must be capable of transferring all of the sounds from the patient to the observer s ear

2 The transferred sounds must possess a quality or pitch of such a character as to be familiar to the observer

3 The stethoscope must possess a means of relatively attentuating certain portions of the auscultatory frequency spectrum in order to reduce masking effects

To obtain a starting point in our effort to ascertain the optimal frequency response of a stethoscope, two of us (M B R and H B S) [10] made a study of the acoustic stethoscopes which had been on the market They were found to vary in tonal quality, and, in addition, the various commercially available

amplifying stethoscopes are being marketed to this day which alter the quality of the auscultatory sounds in the manner described The low pitched, raspy effect is more common in the electrical stethoscopes on the market than in the acoustical varieties because it is easy to accentuate low pitched sounds with an amplifying system whereas, in the acoustical stethoscope, the low pitches are limited in relative intensity by the physical nature of the apparatus If extreme care is not taken by the manufacturer in duplicating the frequency response of the average acoustical stethoscope, the amplifying stethoscope alters the quality of the auscultatory sounds

EFFECTS OF SOUND INTENSITY VARIATION

It has been shown that average human hearing is most sensitive to variations in intensity above 50 decibels and gradually becomes less sensitive at the lower sensation levels This characteristic of hearing is important in auscultation for the intensity level is often 10 decibels above threshold, or less, and the frequency spectrum which is involved is at the lower extremity of the human hearing range It is well to repeat that, with a frequency of 60 cycles per second an intensity variation of 20 per cent is just perceptible when the sound level is 50 decibels, and a variation of as much as 200 to 300 per cent is necessary when the intensity level is 10 decibels above threshold

Although a higher sensation level requires less of a frequency change for minimum perception, the ability of the auditory system to detect changes in frequency at the lower levels of intensity is not so poor as its ability to perceive variations in intensity

It has also been pointed out that the intensity of sounds bears an important relationship to masking pitch and so forth Stevens and Davis,[1] [1] in their discussion of this phase of hearing stated

> It has been the traditional view of psychology that the attributes of sensation show a one to one correspondence to the dimensions of the stimulus Some such view is also implicit in the naive epistemology of the physicist He tends to think of pitch as if it were the perception of the frequency of a tone but we have seen that holders of that view run into difficulties The pitch of a pure tone can be altered without changing its frequency likewise the loudness of a tone may be varied without changing its intensity Pitch is a function of two physical variables frequency and intensity loudness is a different function of the same two variables Both pitch and loudness are fundamentally to be conceived as reactions on the part of organisms to sound waves These are systematic reactions to be sure and can be ordered on scales and evaluated but they are nevertheless products of the interaction of an atmospheric disturbance with a living system

Stevens and Davis further stated

> Several writers during the last century have discussed the fact that tones are characterized by an apparent largeness or extensiveness [1] The low tones of an organ appear to be bigger than the high chirp of a cricket even when the loudness

tion of the characteristic curves of the chest pieces in figure 12) A theoretical frequency response curve for an amplifying stethoscope (not including the modifying effects of the chest pieces, which are capable of reproducing the sounds as they are heard with the theoretically average acoustic stethoscope) is shown in figure 32 The construction of an amplifying stethoscope which has a frequency response characteristic identical with the theoretical one of figure 32 is practical, and this feature has been in

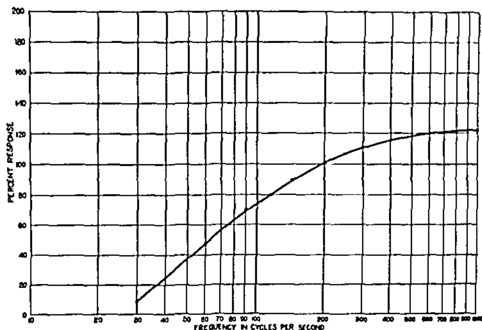


Fig 32 Output of amplifying stethoscope versus input to microphone omitting characteristics of chest piece Ordinate plotted in per cent of response instead of in decibels to accentuate graphically the degree of slope (From Rappaport M B and Sprague H B) [10]

corporated in the apparatus It is well to mention that a slight variation in the slope of the curve below 200 cycles per second is sufficient to alter considerably the quality of the sounds as they are heard with the amplifying stethoscope A variation of considerable degree in the slope of the curve above 200 cycles per second produces little effect on the quality of the auscultatory sounds

An amplifying stethoscope with a frequency response curve which is flatter below 200 cycles per second than the graph of figure 32 has a tendency to convey the sounds to the observer's ears (especially the low pitched, mitral diastolic murmurs) with a raspy effect This foreign quality is so different from what is normally heard with the acoustic stethoscope that at times, the observer has the feeling that the time he spent in perfecting his auscultatory technic was wasted, unless he keeps going back to his acoustic stethoscope This has been the major difficulty with electrical stethoscopes, it is no fault of the system, but of the basic design of the apparatus An interesting fact is that, although we described this phenomenon in 1941,[10]

the individual is not practical except with an amplifying stethoscope which has an adjustable and calibrated intensity control and in which the microphone, amplifier, and audiophone possess fixed sensitivity characteristics

SOUND FILTERS

An acoustic or electrical filter system may be used with the electronic or amplifying stethoscope as an aid in minimizing masking effects Acoustic filtering is accomplished by means of the open bell and diaphragm chest pieces, which can be attached to the microphone Electrical filtering is obtained by inserting a Campbell filter, or any of the well known modifications of the Campbell filter, between the microphone and the audiophone

Dodge and Fredrick[153] first employed the Campbell filter in an amplifying stethoscope The filter was composed of a series of high pass and low pass filters By means of a suitable switching system, any filter or combination of filters could be inserted into the amplifier circuit to allow any desired low medium, or high portion of the auscultatory spectrum to pass to the translating device The amplifier proper had a frequency response characteristic that was flat over the auscultatory range In 1940, Mannheimer described a system similar to that of Dodge and Fredrick and such a system is manufactured by Elma of Stockholm

From the physicist's point of view, the Dodge and Fredrick[153] system is an excellent instrument for analyzing the frequency components of sounds Williams and Dodge[154] and Cabot and Dodge[155] utilized the apparatus as an auscultatory wave analyzer with noteworthy results, and Mannheimer has shown good phonocardiographic results On the other hand, the clinical use of the apparatus is limited by the fact that it alters the quality of the sounds as compared to what is heard with the ordinary acoustic stethoscope The modification of tonal quality occurs because of a deviation from the over all frequency response of an acoustic stethoscope Furthermore the sharp cut off characteristics of the Campbell filters which are desirable when the instrument is used as a sound wave analyzer, produce additional marked modifications to the auscultatory sound effects

For optimal results a stethoscope whether it be electrical or *acoustic*, should possess a frequency response characteristic *similar to that of the* average commercial acoustic stethoscope minus the modifying effect of the chest piece The filters, whether they be electrical or acoustic should possess modifying frequency response characteristics similar to those obtained with open bells of various diameters and diaphragm chest pieces made of plastic materials In other words the function of a filter in a stethoscope is not to eliminate a certain band of sound frequencies but to alter the slope of the over all response of the basic curve shown in figure 32, and relatively attenuate a certain portion of the spectrum

Normally cardiovascular sounds are not pure tones but noises, and are

of the two are equal This subjective aspect of a tone is known as volume (The radio engineer speaks of the volume of a sound, or of the 'volume control of a radio set but he means by volume what we should properly call intensity)

In order to illustrate the manner in which these characteristics of hearing are related to auscultation, let us assume that a certain heart lesion is present in an emaciated person, an obese person, a heavy, "barrel chested" man, and a person with a chest of normal proportions Let us further assume that the intensity, pitch, and quality of the heart sounds and murmurs are identical at the source in each case Because of the different transmissional characteristics in these individuals, the intensity of the sounds, as they are heard on the surface of the chest with the same acoustic stethoscope, varies decidedly As a result of the intensity modification alone, sounds that are audible in the normal and emaciated persons may not be heard at all when the stethoscope is applied to the obese and "barrel chested" patients

If the acoustic stethoscope employed in these cases had an adjustable intensity control, and the intensity levels were the same in all (approximately that of the sounds heard in the patient with a normal chest), a considerable percentage of the distortion, or modification effects, would be eliminated Also, certain possible masking effects could be reduced by slightly varying the intensity above or below that of the sounds heard over the normal chest, a noticeable variation in quality and pitch would also be apparent at the various hearing levels Obviously, the acoustic stethoscope does not lend itself to adjustable intensity control, whereas the amplifying stethoscope is capable of such control

From this discussion it should be obvious that an amplifying stethoscope is not an instrument to be used primarily for making sounds many times louder than they can be heard with an acoustic stethoscope The major advantage of the amplifying stethoscope over the acoustic stethoscope is that the intensity can be adjusted as desired, and thus a number of modifying characteristics which cannot be overcome with the acoustic stethoscope are eliminated The intensity of heart sounds in a normal young person, as perceived by auscultation with an acoustic stethoscope is approximately optimal from a standpoint of masking, pitch, quality, and accustomed usage of the older acoustic stethoscope

Because of deviations in the individual physician's hearing from the "average normal threshold curves of audibility" (Fig 5), it is obvious that, in any given case, the sounds heard by a group of trained observers, employing the same acoustic stethoscope, will not register identically, although the hearing of each may be considered normal The most marked variations from normal are found in the presence of impaired hearing An increase in the sensitivity of the stethoscope, to compensate for the decreased acuity of hearing, brings back the auscultatory keenness of hearing which is so necessary in clinical cardiology At present, such compensation, to suit

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THE LOUD SPEAKER

A loud speaker system * designed to operate with the amplifying stethoscope, is shown in figure 33 [10], it is useful principally for teaching Most

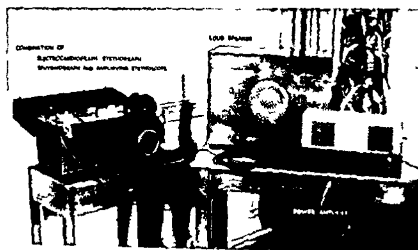


Fig 33 A loud speaker system for auscultatory applications (Courtesy of Sanborn Company Waltham Massachusetts)

loud speaker systems have utterly failed to reproduce sounds of unmodified quality with this one we have been able to produce a fair approximation of the sounds as they are heard with the acoustic stethoscope or the amplifying stethoscope and the audiophone Most attempts at loud speaker reproduction of auscultatory sounds have been poor because of the following

1 The combinations of microphone amplifier and loud speaker did not possess an over all frequency response similar to that of the ordinary acoustic stethoscope

2 Ordinary radio loud speakers cannot reproduce the low frequencies encountered in cardiovascular sounds

3 Invariably the power handling ability of the loud speakers and power amplifiers were insufficient to reproduce the cardiovascular sounds with sufficient intensity and with no overloading distortion

The form of distortion that may be introduced by item 1 has been discussed previously (see p 82) A loud speaker which does not possess a frequency response over the auscultatory spectrum similar to that of the audiophone previously described alters the quality of the heart sounds The average radio loud speaker has a resonance peak of 100 cycles per second, or less As a result, a booming character is imparted to the first and second heart sounds producing a tom tom drum effect This drum effect, in addition to altering the quality of the heart sounds, decidedly masks any systolic and diastolic murmurs that may be present The loud speaker in figure 33

* Manufactured by Sanborn Company Waltham Massachusetts

therefore, composed of a conglomeration of unrelated frequencies of multitudinous intensities Each component frequency performs a distinct function, in that it imparts a distinguishing quality to the cardiovascular sound Although a cardiovascular sound may appear high pitched, wave analysis usually shows that it comprises low, as well as high frequencies, although the higher frequencies predominate In cases where the cardiovascular sound appears to be low pitched, the reverse usually holds true

To minimize the masking effects that a more intense and lower pitched sound may produce on one of higher pitch, as, for example, in the case of the diastolic murmur of aortic insufficiency, a relative attenuating effect on the intense lower frequency components of the second heart sound would reduce the masking and fatigue effects on the auditory system and would make the faint high pitched diastolic murmur appear more distinct For best results with the Dodge and Fredrick filters, using sharp cut off, high pass filters, the most suitable point of cut off depends on the component frequencies of the sounds Considerable variation is found in the relative proportions of high and low frequencies in murmurs of aortic regurgitation On the other hand, a filter such as the Bowles diaphragm chest piece, which progressively attenuates, but does not eliminate, the lower frequencies, is obviously less critical and more suitable for clinical use A modified Dodge and Fredrick filter system with a continuously adjustable cut off filter may be employed Confusion may arise with such a filter because of the difficulty in selecting and reproducing the optimum cut off setting in routine clinical work

In some cases where the aortic diastolic murmur was extremely high pitched and at threshold level, we have been able to register the murmur more distinctly by using a thicker diaphragm (0 035 in) in the Bowles chest piece or by inserting a high pass electrical filter which, additionally, relatively attenuates the low frequencies but does not eliminate them Although the quality of the auscultatory sounds is altered, masking and fatigue effect are reduced in auscultation, and the signal to noise ratio is improved in the phonocardiogram This procedure is of help in determining the presence or absence of a threshold aortic diastolic murmur of insufficiency providing due consideration is given to alterations in the other cardiovascular sounds

Campbell filters, or their modifications may be so designed that they reproduce the frequency response characteristics of open chest pieces of various sizes as well as of diaphragm types, the chest piece with the lowest pitch is employed, and the auscultatory equivalents of the higher pitched chest pieces are obtained by inserting the equivalent electrical filter

The choice between an acoustic filter and an electrical filter therefore reduces itself essentially to a question of economics Obviously, the acoustic filters are less costly and less massive Also, the clinician is more familiar with the acoustic filter system

BONE CONDUCTION TRANSDUCERS

A suitably designed bone conduction transducer may be used in place of the audiophone for converting the amplified electrical pulsations into sounds The partially deaf physician whose hearing is more efficient by bone conduction than air conduction obviously can benefit by this arrangement

THE PHONOCARDIOGRAPH

The manner in which the cardiovascular sounds are graphically recorded on sensitized photographic paper or film has been described (Fig 21) When stethoscopic response is desired in the phonocardiogram, the same electrical pulsations which are applied to the audiophone are applied to the d Arsonval galvanometer and recorded graphically Thus, the cardiovascular sounds may be heard during the process of registration The ability to hear the sounds during registration is most important in good phonocardiographic technic, as correct location of the chest piece, detection of leaks between chest piece and precordium, detection of nonco-operation of patient in the suspension of breathing, and so forth, are essential When "logarithmic" phonocardiograms are registered, it is preferable that the filter not modify the sounds in the audiophone but in the galvanometer circuit only Actually, the human auditory system acts like the logarithmic filter and galvanometer, and if the audiophone should introduce the effects of the logarithmic filter, in addition to the logarithmic effects of hearing, an undesirable auscultatory effect would be produced and an unknown relationship would exist between the sounds as heard and recorded

THE SPHYGMOGRAPH

In certain cases, a simultaneously registered phonocardiogram and electrocardiogram may not supply sufficient information for a complete analysis of all the events of the cardiac cycle An additional simultaneous recording of a sphygmogram and phonocardiogram, or all three together, must be resorted to If two of the phenomena are registered at the same time, the three may be correlated Orias and Braun Menéndez [7] who have done a considerable amount of work on the physiologic aspects and interpretation of phonocardiograms, using the membrane technic, stated as follows

> Investigators who persist in the belief that the electrocardiogram will give all the data necessary for the interpretation of phonocardiograms will find themselves seriously limited in their studies and will not be in a position to make the fullest use of their results

The authors give an illustrative example

> Those workers who have relied on the electrocardiogram alone to localize and correlate the vibrations recorded in the phonocardiogram have been forced to make

is totally free of such resonance peaks in the auscultatory region and is fla in response down to 29 cycles per second

We have observed that a loud speaker system must be capable of handlin a *minimum of 15 watts in order to reproduce the heart sounds without dis*tortion in the average amphitheater or classroom On first thought, so muc power seems unnecessary However, the large excursions of the loud speake diaphragm which are produced by the first and second heart sounds show that a large amount of power is being fed into the loud speaker The reaso *the diaphragm excursions do not produce terrifically loud sounds is tha* human hearing is especially deficient in the frequency spectrum where thes sounds occur

In order to deliver sufficient power to the loud speaker, a power amplifie must be interposed between it and the amplifying stethoscope The amplifie must not modify *the over all frequency response characteristic of the sys*tem, that is, it must possess a flat frequency response over the auscultatory range

Clinical tests have shown that, even with an optimally designed loud speaker system, reproduction of the sounds can only approach the quality as it is heard with an acoustic stethoscope or an amplifying stethoscope and audiophone Room acoustics may produce a modifying effect, and at no time can the closed system of hearing, as is obtained with an acoustic stethoscope or audiophone, be reproduced Thus, the very low pitched cardiovascular sounds are not as well heard via the loud speaker, but the medium and higher frequencies are well reproduced It has also been observed that hearing efficiency improves for the low pitches if the observer comes closer to the loud speaker or listens in a small room It appears that the reason for *this phenomenon is that, at very low auscultatory frequencies, the sense of* auditory feel plays an important part A closed binaural conduction system increases the efficiency of auditory feel for the low frequencies, while there is reduced efficiency of auditory feel with a loud speaker system which must operate in a large volume field

When operating a loud speaker stethoscope, one must be careful of feed back howls or oscillations If the patient is in the same room as the loud speaker, and if the microphone is raised away from the precordium, an oscillatory *howl will occur This oscillation occurs as a result of an acoustic* reaction between the microphone and the loud speaker which builds up into an acoustic howl Also if the patient is placed too close to the loud speaker, the patient's chest may act like a sounding board by picking up the sounds from the loud speaker, feeding them into the microphone, and producing the feed back howl

It is our opinion that loud speaker reproduction is useful only when a large group needs to listen for gross changes in sound quality and for the detection of *arrhythmias For critical listening in group auscultation, multi*ple audiophones are better

mental, determines the configuration of the sphygmogram The highest harmonic frequency of importance in sphygmograms is about the tenth Therefore in the case of a moderately rapid heart rate of 120 beats per minute, where the fundamental frequency is two cycles per second, the frequency of the tenth harmonic is twenty cycles per second Well designed electrocardiographic amplifiers have flat frequency response characteristics up to at least forty cycles per second, and the sphygmographic transducer must be as good or better, in the upper end of the frequency response characteristic to produce an accurate sphygmogram

The combination of crystal and its foil electrodes with no pressure or twist applied may be considered a condenser with a finite capacity When pressure or twist is applied the crystal acts like a generator with a condenser in series The electrical energy which is developed in a piezoelectric crystal is extremely small, although the voltage generated may be of fair magnitude

For all practical purposes, the electrical loading (grid resistance) which the amplifier presents to the crystal may be considered as purely resistive, and the voltage which is delivered to the amplifier (across the grid resistor) is equal to the voltage generated by the crystal minus the voltage drop across the effective capacity of the crystal The attenuation which may be produced on the low frequency end of the sphygmographic spectrum by the effective capacity of the crystal is dependent on the frequency of the stimulus and on the relative values of the crystal capacity and the amplifier input resistance The product of the crystal capacity and the input resistance of the amplifier is the time constant in seconds if the capacity is expressed in microfarads and the resistance in megohms

To avoid attenuation of the low frequency components contained in the sphygmogram it is necessary that the time constant be at least 2 5 sec A well designed electrocardiographic amplifier possesses a time constant of at least 2 5 sec or of equivalent performance By equivalent performance is meant that some amplifier systems possess circuitry which introduces a form of compensation with a shorter time constant, but the over all response to the low frequencies is unaltered Some of the commercially available electrocardiographs utilize this amplifier principle

The capacity of a piezoelectric crystal element which can be used in a sphygmographic transducer is equal to approximately several thousandths of a microfarad To attain a time constant of approximately 2 5 sec or more, the resistance in the grid circuit of the amplifier would have to approach impractical values To circumvent this condition, it is imperative to shunt the crystal with a large condenser[161] The resultant sensitivity is thus markedly reduced but, with proper design, adequate sensitivities are possible with the amplification available in an electrocardiographic amplifier, without increasing the grid resistance from nominal values used in electronic electrocardiographs The upper frequency limit of the transducer is equal

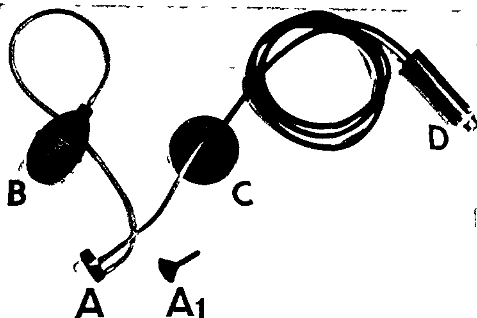


Fig 34 Piezoelectric sphygmographic attachment A Vacuum applicator cup A_1 Pressure type applicator cup B Rubber bulb to produce vacuum in suction compartment of vacuum applicator cup C Piezoelectric transducer D Cord and removable connector which fits into the input receptacle of the electrocardiographic channel of the instrument (From Rappaport M B and Sprague H B) [10]

tubing, serves as a suction chamber for fastening the cup to the patient A pressure type of funnel shaped applicator (A) may be substituted when desired

The pulse sets up air pressure changes in the applicator cup The pressure changes are then transmitted through the rubber tubing to the piezo electric transducer, which converts them into equivalent, minute, electrical pulsations which are, in turn, fed into the electrocardiographic amplifier and recorded like an electrocardiogram

The piezoelectric transducer operates on a principle identical with that of the piezoelectric heart sound microphone previously described (see p 69) However, certain electrical properties are different in the sphygmo graphic transducer to make it suitable for sphygmography

The wave form of a sphygmogram is basically of low frequency That is, the fundamental frequency is equal to the heart rate If the heart rate is 60 beats per minute, the fundamental frequency is one cycle per second and if the heart rate is 120 beats per minute, the fundamental frequency is two cycles per second In a case of bradycardia where the heart rate is for example, 30 beats per minute, the fundamental frequency is one half cycle per second Electrocardiographic amplifiers possess flat frequency response characteristics down to at least one third of a cycle per second, and the sphygmographic transducer must possess the same characteristics

A sphygmogram is not a sine wave, but is composed of a fundamental sine wave with harmonics The harmonic content, when added to the funda

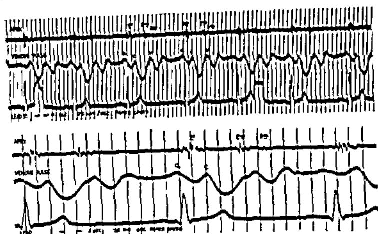


Fig 37 Simultaneously registered phonocardiogram (apex) venous pulse tracing and electrocardiogram (Lead 2) at camera speeds of 25 and 75 mm per second respectively (From Rappaport M B and Sprague H B)[30]

Figure 36 shows a venous pulse tracing which was taken simultaneously with the phonocardiogram for a normal person

The addition of another electrocardiographic channel to the phonocardiograph electrocardiograph permits registration of three simultaneous cardiovascular events Thus, the following combinations are made possible (1) phonocardiogram sphygmogram and electrocardiogram, (2) phonocardiogram and any two sphygmograms, (3) phonocardiogram and any two electrocardiographic leads Other transducers such as manometric electrokymographic or ballistocardiographic may be employed on the electrocardiographic channels

When more than two registrations are made simultaneously, it is advisable

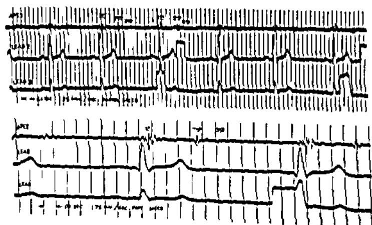


Fig 38 Phonocardiogram (apex) taken simultaneously with electrocardiographic Leads 2 and 3 at camera speeds of 25 and 75 mm per second



Fig 35 Response characteristic of the electrocardiograph channel (From Rappaport M B and Sprague H B)[10]

to several hundred cycles per second In practice, the electrocardiographic channel is usually the limiting factor in the upper frequency ranges

The following are some of the more important characteristics of a piezo electric sphygmograph when it is used in conjunction with an electronic electrocardiograph

1 The speed of response is limited by the d Arsonval galvanometer The galvanometer employed in the original electrocardiographic channel * which we used had a deflection time of approximately 0 01 sec as may be seen in figure 35 The graph was obtained by applying the calibration potential of one millivolt to the input of the amplifier Each interval between adjacent vertical time lines is equal to exactly 0 04 sec A deflection time of 0 01 sec is ample for sphygmographic and electrocardiographic purposes The deflection time in a later design † is equal to 0 001 sec

2 The decay characteristic of the amplifier system is also illustrated in figure 35 For more than 0 2 sec there is no decay whatsoever and thereafter the decay is gradual The flat top characteristic is produced by electrical compensation previously mentioned The 0 2 sec during which there is no decay is longer than the duration of any known electrocardiographic complex and inasmuch as the decay is slow after the 0 2 sec is ample for sphygmographic recording That is the minimal time constant requirements are exceeded by a sufficiently wide margin

3 The electrical sensitivity control of the electrocardiograph is simple to use and it does not alter any characteristic other than the sensitivity of the apparatus

4 Once the recording beam is set slight changes in the adjustment of the apparatus or patient will not result in loss of the beam for it gradually drifts into position because of the decay characteristic of the amplifier

5 In the circuit there are no delicate parts which require protection

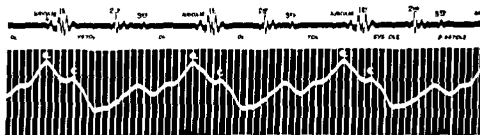


Fig 36 Phonocardiogram and venous pulse tracing of a normal person registered simultaneously (From Rappaport M B and Sprague H B)[10]

* The Sanborn Stetho Cardiette and Tri Beam Waltham Massachusetts

† The Sanborn Twin Beam Waltham Massachusetts

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to employ a white background and record in black, as this allows the three beams to cross each other Figure 37 shows a phonocardiogram, a venous pulse tracing, and an electrocardiogram recorded simultaneously, using this oscillographic technic Figure 38 illustrates a simultaneously registered phonocardiogram and two electrocardiographic leads

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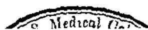
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7 / Graphic Registration of Normal Heart Sounds

PHONOCARDIOGRAPHIC CHARACTERISTICS

Prior to 1941 little consideration had been given to the manner in which phonocardiographic measurements are related to the over all frequency response of the phonocardiograph In 1941 and 1942, two of us[1] suggested that three distinct types of phonocardiographic registration be used namely, (1) linear, (2) stethoscopic, and (3) logarithmic

In the linear registration system sounds which are detected at the surface of the chest and which have a frequency of from zero to more than 1 000 cycles per second—the heart sound vibratory frequency band or spectrum—are neither attenuated nor accentuated with respect to frequency The physicist commonly terms this a 'flat response characteristic" Thus, *linear phonocardiography registers graphically the mechanical vibrations set up by cardiac activity as they exist on the surface of the patient s chest, that is the deviation of the recording galvanometer beam is proportional to the intensity of the vibration at the surface of the chest* Obviously in such a graph the large intensity low frequency vibrations must be controlled by reducing the over all sensitivity of the system for their amplitude may be 10 000 times as great as that of the low intensity high pitched murmurs Therefore in such a system high pitched vibrations often are not registered at all

The stethoscopic system of phonocardiographic registration does not register the cardiovascular sound vibrations as they exist on the surface of the patient s chest but as they are presented to the ears of an observer by means of an average acoustic stethoscope By 'stethoscopic effect' is meant the modifying effects that are introduced at different frequencies by the stethoscope tubing and binaural ear pieces but in each phonocardiogram there is the added effect of the chest piece used in the registration The over all frequency response of the stethoscopic system (Fig 59) is not flat as it is in the so called linear phonocardiogram, but possesses a rising characteristic with respect to frequency increase over the heart sound spectrum—that is the higher the pitch of the sound the better it is registered

The logarithmic system of phonocardiographic registration possesses an over all frequency response equivalent to the summation of the curve of the average human audiogram (Fig 5) and the curve of the average acoustic

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Figure 40 shows how the simultaneous registrations are made Figure 40B is a photograph of the chest piece employed for the simultaneous registration of the stethoscopic and linear, or logarithmic and linear, phonocardiograms This chest piece employs a double outlet to two distinct types of microphones or transducers (Fig 40A), and thus permits simultaneous registration on the same area of the chest

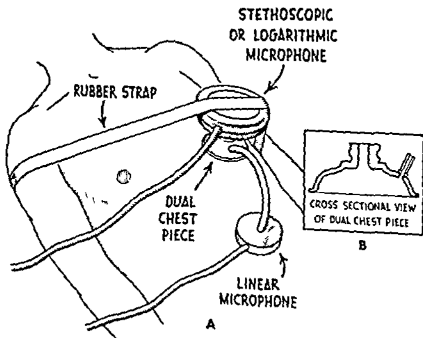


Fig 40 A Arrangement for the simultaneous registration of the stethoscopic and linear or logarithmic and linear phonocardiograms B Cross section of chest piece The chest piece is a modified large or medium open bell shown in figure 10 (From Rappaport M B and Sprague H B)[2]

The linear phonocardiogram of figures 39A and B may readily be recognized as the registration of the "apex beat" or "cardiogram" Wiggers'[3] explanation of the mechanism of the apex beat follows

During ventricular contraction all diameters of the heart decrease the base is pulled downward and the large vessels stretched but the apex does not move upward Owing to the spiral arrangement of the muscle fasciculi the heart rotates to the right with the result that more of the left ventricular surface has a frontal exposure Such rotation particularly affects the apex the forward motion pressing it more firmly against the chest wall This gives rise to the pulsation in the fifth intercostal space referred to as the apex beat Records of this pulsation called cardiograms represent only heart movement

The literature on the cardiogram is sparse[3,9] Wiggers[3] stated

Optical tracings of the apex beat have so far been shown of value in only one respect namely that they incorporate the heart sounds and so allow an exact establishment

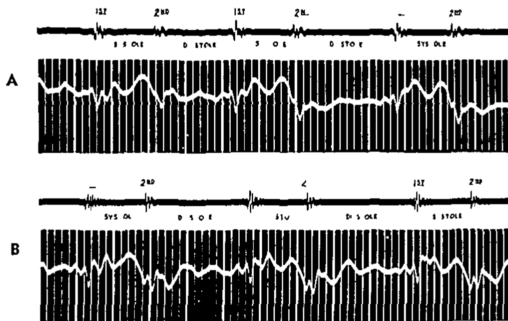


Fig 39 A Simultaneous registration of stethoscopic (upper) and linear (lower) phonocardiograms for a normal person B Simultaneous registration of logarithmic (upper) and linear (lower) phonocardiograms for the same person (From Rappaport M B and Sprague H B)[2]

stethoscope (Fig 32) Thus, the resultant logarithmic phonocardiogram is a graphic representation of the cardiac sound vibrations as they are perceived by the average observer of normal hearing when an average acoustic stethoscope is employed The term logarithmic is used because the shape of the human audiogram in the heart sound auscultatory spectrum approximates a logarithmic slope The resulting phonocardiogram can be considered a "human audiographic" record in which the amplitudes of the component frequencies are registered in proportion to their relative loudness That is, if the first heart sound, as heard, is twice as loud as the second heart sound, the amplitude of the graph of the first heart sound will be twice as great as that of the second heart sound

An understanding of this principle is fundamental to the interpretation of all phonocardiograms *Briefly, it indicates that "loudness" and "intensity" are different factors, and that "loudness" is an auditory perception determined by "intensity" and "frequency" If we attempt therefore, to register what the ear terms "loudness," we must distort our simple linear phonocardiogram by introducing the peculiar logarithmic response of average human hearing and the specific modifications introduced by the average acoustic stethoscope*

Let us now compare the phonocardiograms taken on a normal person at the apex with the linear, stethoscopic, and logarithmic registration systems Figure 39A shows simultaneous registration of the stethoscopic and linear phonocardiograms Figure 39B shows simultaneous registration of logarithmic and linear phonocardiograms

The first heart sound

In the apex cardiogram the first heart sound is usually represented by a few coarse vibrations The stethoscopic phonocardiogram on the same person shows somewhat similar coarse vibrations although attenuated, and some superimposed finer vibrations The logarithmic phonocardiogram greatly attenuates the coarse vibrations *and brings out a larger number of* superimposed, finer vibrations

Various investigators have shown that four factors are responsible for the *production of the major vibrations* which constitute the first heart sound A discussion and bibliography on the subject may be found in the monograph by Orias and Braun Menendez[11] The four factors are (1) the residual vibrations caused by atrial activity—the atrial factor, (2) the muscular contraction and tension of the ventricular walls—the muscular factor (3) the closure of the atrio ventricular valves—the valvular factor and (4) the movements and distension caused by the ejection of blood from the ventricles into the arteries—the vascular factor vibrations caused by opening of the semilunar valves initiate and fuse with this element of the first heart sound In spite of conflicting experimental evidence it appears likely that contraction of the ventricles produces vibrations in the absence of valve closure These constitute the muscular factor and normally contribute to the early part of the first heart sound

Caeiro and Orias[1] *in 1937 indicated that it is possible to distinguish* four distinct components in the phonocardiographic representation of a normal first heart sound We confirmed their observations in 1942, and so did Luisada Mendoza and Alimurung[13] in 1949 *In figure 41, the four*

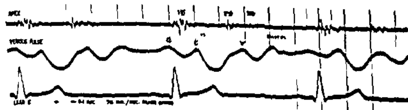


Fig 41 Simultaneous stethoscopic phonocardiogram, venous pulse tracing and electrocardiogram to illustrate the relationship of the four components of the first heart sound (From Rappaport M B and Sprague H B)

components may be distinguished in the stethoscopically registered phonocardiogram The first component is a characteristically low frequency coarse vibration *which commences* with the electrocardiographic QRS complex and normally precedes the apex of the QRS wave When an atrial sound is present the first component usually merges with the atrial sound without a definite interval This phenomenon *may be seen* best in a linear phonocardiogram (Fig 39A) The first component was thus believed to be related to the atrial sound as a residual vibration, or the ' atrial factor

of systole and diastole According to Weber[7] they enable us to determine the beginnings of auricular systole ventricular systole systolic ejection and the opening of the atrioventricular valves and are further particularly valuable in determining the isometric contraction phase It is quite obvious however that the same temporal relations may be established as well by the use of optical venous and arterial pulses Whether any other significance may be attached to pathologic tracings further investigation alone can determine Weitz[8] it is true has published a large series of optically recorded cardiograms both from normal individuals and from those with cardiac lesions Aside from the addition of a variety of murmur vibrations associated with the particular lesions in question and the greater predominance of positive waves they show nothing that can be regarded as distinctive in a diagnostic sense

Taquini, Massell, and Walsh[10] have shown the usefulness of the cardiogram in the differentiation between the opening snap of the mitral valve and the third heart sound when the isometric relaxation phase of the left ventricle is shortened in the presence of mitral regurgitation The authors claim as follows

Under such circumstances the third sound which is produced during rapid inflow into the left ventricle might not show its usual relation to the *v* wave of the venous pulse for the latter is associated with pressure changes in the right ventricle Therefore in order to ascertain the nature of the extra sound in these cases it is necessary to take the phonocardiogram simultaneously with the records of the apical pulsation

THE ATRIAL SOUND

By interrelating the linear stethoscopic, and logarithmic phonocardiograms of figure 39A and B, we find that the cardiogram registers distinctly the sound or vibration associated with atrial systole The stethoscopic phonocardiogram shows a faint atrial vibration and the logarithmic registration does not show any distinct vibrations which represent atrial systole In a *considerable percentage of normal persons the vibrational frequency of a* transmitted atrial contraction is so low that it is not capable of passing through the average acoustic stethoscope When such is the case the stethoscopic phonocardiogram does not register the atrial sound Human auditory perception of an atrial sound of such low frequency and intensity is an obvious impossibility because of the added logarithmic attenuation of human hearing For the same reason, the logarithmic phonocardiogram attenuates and in this case does not register the atrial sound The linear phonocardiogram is the only reliable phonocardiographic means *of indicating the presence* of an extremely low frequency atrial vibration In phonocardiography therefore, the cardiogram is useful as an indicator of the true location of the atrial contraction in the cardiac cycle

The electrocardiogram is a crude reference for evaluating the atrial sound as the electrocardiographic P wave precedes the phonocardiographically registered atrial vibration or sound The venous pulse is a good reference for timing atrial systole as the venous pulse *a* wave is usually synchronous Intra atrial pressuregrams are *the preferred reference* tracings if attainable

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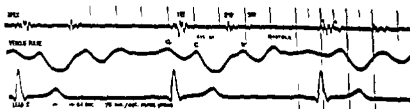


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recognized in phonocardiograms, following the P wave of the simultaneously registered electrocardiogram and preceding the onset of the QRS complex, and that such vibrations maintain their time relationship with the P wave when the P R interval changes However, the initial low frequency, low amplitude vibrations of the first heart sound that succeed the beginning of the QRS do not bear such relationship to atrial systole, occurring even in its absence On the other hand, they do show a constant relationship to the main vibrations of the first heart sound, and if we accept, as the evidence would indicate, that the beginning of the *main* vibrations (that is, the second component) of the first sound marks closure of the atrioventricular valves, then the initial low frequency, low amplitude vibrations (that is, the first component) occur during the preisometric phase of ventricular systole Again the initial group of vibrations coincides with, or follows immediately after, the beginning of the systolic impulse of the apex cardiogram These observations are consistent with the earlier suggestion of Wiggers[15] that "when the ventricle begins its contraction its position changes slightly This may produce the first feeble vibrations of the first heart sound" In 1940 Wiggers and his co workers[16] accepted the explanation of Caeiro and Orias[1] concerning the atrial origin of these pre isometric vibrations of the first heart sound However Wiggers pointed out that while these sounds begin definitely before the rise of intraventricular pressure they may continue slightly into the early period of rising tension, and those vibrations which do continue represent initial vibrations of the first ventricular sound rather than the termination of the atrial sound[15]

It is apparent that the initial vibrations shown in figure 42 occur with the beginning of ventricular contraction and truly constitute the first component of the first heart sound They are associated with the development of tension in the ventricular muscle fibers which initiates the rise of intraventricular pressure Since these vibrations occur before the main group associated with closure of the atrioventricular valves they must be due directly to (1) slight shortening of the muscle fibers initiating the rise of intraventricular pressure (2) the taking up of slack in the valve leaflets before they become opposed or (3) conceivably to movement of the heart within the thorax

In the majority of instances vibrations of atrial origin which precede the beginning of ventricular contraction, can and should be differentiated from the first heart sound and the term "first component of the first heart sound" should include only the initial low frequency vibrations associated with the beginning of ventricular systole

The vibration which comprise the second component may be distinguished from the vibrations of the first component by their characteristically higher frequency and larger amplitude in a stethoscopically or logarithmically registered phonocardiogram The second component always commences after the apex of the electrocardiographic QRS complex and represents the beginning of the isometric, or presphygmic phase of ventricular contraction

In 1951, Counihan and his co workers,[14] on the basis of further observa tions, suggested a modification to the interpretation of the first component of the first heart sound They observed that in phonocardiograms recorde at the cardiac apex of 28 patients having atrial fibrillation and 2 havin complete heart block, vibrations of low frequency and low amplitude we present at the beginning of the first heart sound in every person havin atrial fibrillation (Fig 42, bottom) In the 2 patients who had comple heart block, even when atrial systole preceded ventricular systole by a interval sufficient for the atrial sound to have expended itself, the initi vibrations were still present (Fig 42, top)

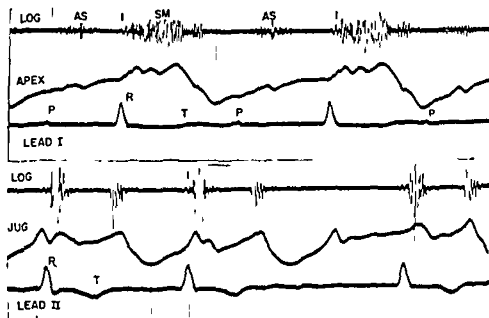


Fig 42 Phonocardiograms recorded with logarithmic response Top Complete hear block Heart sounds at apex with apex cardiogram and electrocardiogram AS = atria sound SM = systolic murmur The numeral I refers to the first component of the first hear sound Bottom Atrial fibrillation Heart sounds at apex with jugular pulse and electrocardio gram The numeral I refers to the first component of the first heart sound (From Counihan T B Messer A L Rappaport M B and Sprague H B)[14]

The average duration of this group of vibrations was 0 05 sec, with a range of 0 03 to 0 06 sec, and the average fundamental frequency was 35 cycles per second, with a range of 20 to 60 double vibrations Although the stethoscopic phonocardiogram registers low frequency vibrations more ef ficiently, the initial first heart sound vibrations lie within the frequency range of human audibility and are well registered with logarithmic response, as may be observed in figure 42 The initial vibrations may be seen to com mence after the beginning of the QRS complex in the electrocardiogram, which is recorded simultaneously, and with, or shortly after, the beginning of the rise of the first positive systolic wave in the apex cardiogram

There is no question that vibrations of atrial origin frequently can be

ng adds a still greater degree of selective attenuation An interesting fact is that, often, evidence of a first and fourth component may be present in the logarithmic phonocardiogram but the observer, although extremely competent will not hear the lower frequency vibration because of masking effects[1] of the apparently more intense second and third components

Simultaneous registration of the stethoscopic or logarithmic phonocardiogram with the venous pulse tracing may serve as a means of differentiating between a prolonged first heart sound and a first heart sound followed by a short systolic murmur None of the four components of the first heart sound normally has a duration which allows it to extend beyond the peak of the venous pulse *c* wave *Thus, it is reasonably safe to say that vibrations which extend beyond the peak of the c wave are the components of a systolic murmur* On rare occasions an extremely low frequency wave may occur just after the peak of the *c* wave (during reduced ventricular ejection) Also the fourth component vibrations are characteristically of a lower frequency than are those of a systolic murmur which also aids in the differentiation even though the systolic murmur may commence early in the first heart sound, but not before the isometric contraction phase, that is the second component

Kountz Gilson Smith and Edwards[17,19] in 1941 described 'vibrograms' of the heart sounds with an apparatus which had a drop-off in transmission of less than two per cent between five cycles and fifteen hundred cycles per second The authors gave no figures for frequency response below five cycles per second If the over all response was flat from practically zero cycles per second the apparatus was equivalent to the linear phonocardiograph and could be classified as a good sphygmograph If the vibrograph response dropped sharply below five cycles per second it would no longer be an accurate sphygmograph but could be classified as a phonocardiograph which is linear between five cycles and fifteen hundred cycles per second They used cathode ray recording which does not offer additional advantages over mirror or d Arsonval oscillographic procedures such as employed herewith The major utility of cathode ray oscillography is in the registration of supersonic and radio frequencies which are not present in cardiovascular sounds Actually it is much more difficult to obtain satisfactory recording definition with a cathode ray system than it is with an optical galvanometric system In 1951 Johnston[16] made similar studies with a manometric system and shortly thereafter Luisada and Magri[1] used the linear phonocardiographic system we have described and observed that the linear phonocardiographic system gave essentially the same results as Johnston's

THE SECOND HEART SOUND

In 1832 Billing suggested that the second heart sound may be attributed to the closure of the semilunar valves In the same year Rouanet[3] set up an experiment and verified Billing's theory In 1882 Webster[4] sug

The second component records the closure of the mitral and tricuspid valves

Usually, the third component can be distinguished from the second (although it is somewhat similar in appearance) by a separation or slight splitting effect *The third component registers at the time of the opening of the semilunar valves and the onset of ventricular ejection* It is difficult to be certain whether this component is due to semilunar opening or tricuspid closure

The fourth component is composed of coarse vibrations which may extend to the peak of the c wave of the venous pulse or the anacrotic notch of a simultaneously registered central arterial pulse Often the fourth phase vibrations terminate prior to, but rarely after, the peak of the c wave in a normal person Thus, *the vibrations which comprise the fourth phase are believed to be produced by the acceleration of the blood in the arteries during the maximum ejection phase of ventricular systole*

In the linear phonocardiogram, there is always a preponderance of low frequency vibrations and a total, or almost total, lack of any of the higher frequency components that are so obviously registered in the stethoscopic and logarithmic phonocardiograms A harmonic analysis of the frequency components of the first heart sound, which is a conglomeration of unrelated frequencies, shows that the coarse, or low frequency, vibrations are of much greater intensity than are the higher frequency components *The amplitude ratio of the major coarse and fine vibrations is so large that, when registered linearly, the fine vibrations are entirely lost or are, at best, insignificant Thus, the linear phonocardiogram is extremely efficient in the registration of the first and fourth phases of the first heart sound and decidedly inefficient as an indicator of the second and third phases*

The stethoscopic phonocardiogram attenuates the first and fourth phases of the first heart sound and brings out distinctly the higher frequency second and third components The reason the second and third components appear to be of such magnitude in this type of registration is that more amplification is employed than is permissible in linear phonocardiography If an equal amount of amplification were employed in linear phonocardiography, with the object of bringing out the second and third components the deflections of the first and fourth components would be so large as to mask the second and third components *With stethoscopic registration, and the additional amplification the attenuation effects on the first and fourth components are not sufficient for obliteration, but are sufficient to bring out clearly the second and third components*

Although the average acoustic stethoscope is capable of conducting the selectively attenuated four components of the first heart sound to the tympanums of the ears, the observer is capable of hearing merely the higher frequency second and third components and little or practically none of the first and fourth components The reason human perception is so inefficient in the detection of the first and fourth components is that logarithmic hear

third component distinctly The fourth component is frequently present but considerably attenuated in a stethoscopic phonocardiogram In figure 43, a small wavelet representing the fourth component can be seen

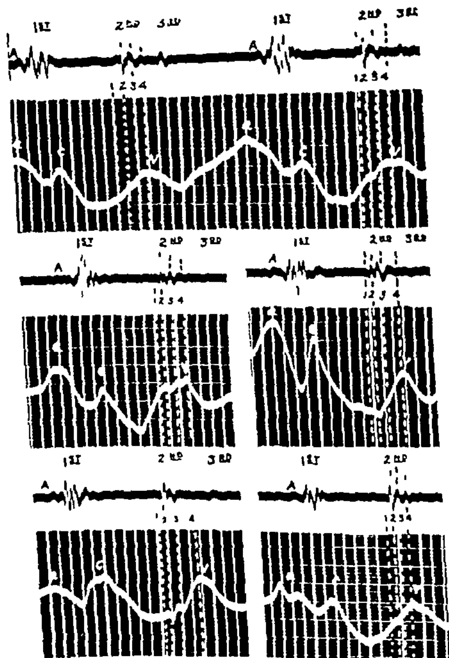


Fig. 44 S m ltaneous st thoscop c ph no ard ograms at apex (pp r) and venous puls tr c ngs (lower) of Fv no mal p r ons show ng the fo r components of the second heart sound and th r lat onsh p to the e o s p lsations The atr al f st and third heart sounds e e also r g stered d st ctly (F m Rappapo t M B a d Sprague H B)2

gested that additional sound vibrations may occur during this phase of cardiac cycle as a result of vibrations of the arterial wall and blood colu The consensus at the present time favors these investigators

Graphic analysis of a large number of phonocardiograms has shown t the composite, normal, second heart sound, as recorded at the apex, is co posed of four distinct components[2] The reason a composite of a large nu ber of normal graphic registrations of the second heart sound is refer to in the analysis is that all four components do not always register d tinctly in a single phonocardiogram Figure 43 shows a simultaneous stet scopic and linear phonocardiogram, registered at the apex, in which the fo components may be detected In figure 44, there are additional phonocard grams which show the four components of the second heart sound

A composite linear phonocardiogram shows that the second heart sou commences with a small, subaudible vibration which we call the first co ponent The first component is distinct in the linear phonocardiogram figure 43 This initial vibration is immediately followed by a few coarse v brations—the second component In figure 43, the second component is re resented by a large, notched vibration The second component is, in tur followed by one or more coarse vibrations which are usually of smaller i tensity than those of the second component In figure 43, the third com ponent is represented by a single vibration At the end of the thi component, and simultaneous with the apex of the *v* wave of a venous pul registration, a coarse vibration, or negative dip, terminates the second hea sound—the fourth component

A composite, normal, stethoscopic phonocardiogram shows the first com ponent of the second heart sound, but with some attenuation, this is we represented in figure 43 The first component is more distinct in some of th records of figure 44 The second component is present, with the addition o superimposed higher frequency components, this is well illustrated in fig ure 43 The third component is present, but attenuated, figure 43 shows th

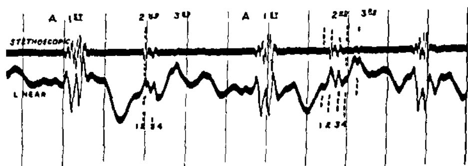


Fig 43 Simultaneous stethoscopic (upper) and linear (lower) phonocardiograms at the apex of a normal person The four components of the second heart sound are present in the stethoscopic phonocardiogram and their temporal relationship to the apex cardiogram is shown A slight atrial vibration and the first and third heart sounds are registered distinctly

and tendinous structures or valves Furthermore, though this cause is stressed as the principle one, other subsidiary factors are not necessarily excluded Some authors[33, 34] stress the importance of an increased cardiac distensibility (diminution of tone) due to vagotonic disturbances favoring the production of the sound"

Almost every linear phonocardiogram shows some evidence of a low frequency vibration which starts during the downslope of the simultaneously registered venous pul e 1 wave In some normal persons a single positive vibration may be present and, in others, two or possibly three such vibrations appear but are of lesser intensity The stethoscopic phonocardiogram usually shows the third heart sound as a single oscillation but not so frequently or distinctly as does the linear phonocardiogram The logarithmic phonocardiogram likewi e registers a normal third heart sound as a single oscillation Registration of a normal third heart sound is decidedly less frequent in a logarithmic phonocardiogram

THE DURATION OF THE FIRST NORMAL HEART SOUND

Table 7 is a compilation of data found in the literature pertaining to the duration of the first heart sound in normal persons The minimum normal duration of the first heart sound as observed by Roos was 0 041 sec, whereas Strahl claimed that it was 0 125 sec—approximately a 3 1 ratio

TABLE

COMPILATION OF DATA FOUND IN THE LITERATURE PERTAINING TO THE DURATION OF THE FIRST HEART SOUND IN NORMAL PERSONS

Author	Year of Observation	Duration of First Sounds in Seconds	Method
Einthoven W [3]	190	0 058 0 1 6	Electrical
Roos E [36]	1908	0 041-0 064	Flame
Weiss O [37]	1909	0 068	Direct
Gerhartz H [38]	1911	0 11	Direct
Kahn R H [39]	1911	0 095-0 109	Electrical
Lilien tein [40]	1911	0 09	Electrical
Ohm R [41]	1912	0 083-0 09	Direct
Ev ter J A E [4]	1911	0 128	Electrical
Kapff W [43]	1914	0 0 9-0 116	Direct
Brid man E W [44]	1915	0 145	Electrical
Strahl E O [4]	1920	0 125 0 1 5	Direct
Kanner L [46]	1921	0 16	Direct
Wiggers C J [47]	1923	0 05-0 152	Direct
Schutz E [8]	1933	0 09-0 12	Electrical
Orias O [4]	1936	0 10 0 17	Direct
Segura A [49]	1937	0 10-0 14	Direct

From Rappaport M B and Sprague H B -

A composite logarithmic phonocardiogram shows the presence of a first component of the second heart sound. The second component is present with more superimposed higher frequency vibrations than appear in the composite stethoscopic phonocardiogram. Usually, the third and fourth components are completely eliminated.

The beginning of the second heart sound, or the first component, occurs at the beginning of the relaxation and diastolic fall of pressure in the ventricle. The second component vibrations may be ascribed to the closure of the semilunar valves. The vibrations that comprise the second component contain many frequencies, among which the lowest predominate. This accounts for the introduction of higher frequency components in the stethoscopic phonocardiogram and still higher components in the logarithmic phonocardiogram, at the expense of the lower frequency vibrations.

The third component of the second heart sound is probably caused by vibrations of the arterial wall and blood column. An additional source of vibration in this region may be the natural period vibration of the chest wall, which may be set into oscillation by the vibrations of the second component.

The fourth component is the result of the opening of the mitral and tricuspid valves, and it occurs simultaneously with the apex of the venous pulse *v* wave. The common occurrence of the fourth component in a considerable percentage of linear and stethoscopic phonocardiograms from normal persons, and its total absence in logarithmic phonocardiograms, indicate that the so-called "opening" snap is present in normal persons at a subaudible level. Only in mitral stenosis does the fourth component become audible.

In every normal phonocardiogram the train of vibrations which comprise the four components of the second heart sound terminates with, or prior to the occurrence of the simultaneously registered venous pulse *v* wave. Thus a simultaneous phonocardiogram and venous pulse tracing should supply sufficient data for the differentiation between a prolonged second heart sound and a second heart sound followed by a short diastolic murmur. *In other words any vibrations which continue beyond the apex of the simultaneously registered venous pulse v wave are components of a diastolic murmur. This of course excludes the vibrations that may be set up by a third heart sound.*

The third heart sound

The generally accepted explanation of the cause of the third heart sound is well summed up by Orias and Braun Menendez.[11] "The third heart sound is caused by vibrations of the ventricular walls due to their sudden distension by the inrush of blood from the auricles in the first moments of rapid ventricular filling." According to Orias and Braun Menendez, this third heart sound hypothesis was first proposed by Ohm[5] in 1921, and has been supported by many authors."[8, 33] Orias and Braun Menendez further stated "By the term 'ventricular walls' no distinction is implied between muscular

TABLE 8

DURATION OF THE FIRST HEART SOUND IN 33 NORMAL PERSONS MEASURED IN STETHOSCOPIC AND LOGARITHMIC PHONOCARDIOGRAMS

Case	Age in Years	Duration of First Heart Sound (Seconds)	
		Stethoscopic	*Logarithmic*
1	24	0.160	0.170
2	24	0.120	0.090
3	28	0.120	0.090
4	22	0.135	0.120
5	21	0.125	0.105
6	22	0.140	0.120
7	19	0.130	0.100
8	21	0.160	0.115
9	20	0.145	0.125
10	26	0.105	0.080
11	23	0.155	0.125
12	24	0.105	0.100
13	22	0.140	0.115
14	22	0.120	0.110
15	21	0.150	0.115
16	22	0.110	0.095
17	22	0.115	0.080
18	21	0.110	0.085
19	21	0.110	0.090
20	22	0.150	0.135
21	21	0.170	0.090
22	20	0.115	0.095
23	23	0.125	0.100
24	22	0.165	0.105
25	22	0.150	0.120
26	21	0.130	0.105
27	22	0.120	0.110
28	22	0.140	0.085
29	19	0.110	0.100
30	23	0.110	0.095
31	21	0.115	0.105
32	38	0.115	0.085
33	36	0.120	0.085

From Rappaport M B and Sprague H B [2]

is highly efficient in the low frequency region where the normal heart sounds lie Also the application pressure [1] with such a chest piece is not critical (The smaller the diameter of the chest piece the more critical is the appli cation pressure to the patient s chest with respect to low frequency attenu ation)

Smaller diameter chest pieces and certainly the Bowles diaphragm types, attenuate the first and fourth components to a greater extent than does the

Einthoven's measurement for maximum normal duration was 0 176 sec, whereas Roos' was 0 064 sec —1 2 75 1 ratio

Differences in maximum and minimum measurements cannot be wholly attributed to the phonocardiographic method employed (such as direct versus electrical) As shown in table 7, among the authors who employed the direct method, Wiggers (0 05 sec) and Strahl (0 125 sec) showed the largest difference for minimum duration of the first heart sound—a 2 5 1 ratio The greatest difference for maximum duration of the first heart sound was between Strahl (0 175 sec) and Ohm (0 097 sec)—a 1 8 1 ratio

Similar differences in measurement existed among the investigators who employed the electrical phonocardiographic method Einthoven obtained a minimum duration for the first heart sound of 0 058 sec, whereas Kahn observed a minimum duration of 0 095 sec—a ratio of 1 64 1 Einthoven obtained a maximum duration of 0 176 sec, whereas Kahn's measurement was 0 109 sec—a ratio of 1 62 1

A comparison of the data obtained with Einthoven's electrical method and Wiggers' direct method indicates agreement within a reasonable percentage as to maximum and minimum duration

For comparison with the data of table 7, we selected 33 normal, male, university students and made the following registrations at the apex with the student in a supine position [2]

1 Stethoscopic phonocardiogram simultaneous with electrocardiographic lead 2
2 Stethoscopic phonocardiogram with venous pulse tracing
3 Logarithmic phonocardiogram simultaneous with electrocardiographic lead 2
4 Logarithmic phonocardiogram with the venous pulse tracing

Table 8 shows the measurements of the duration of the first heart sound in the 33 normal persons *The maximum duration of the first sound by the stethoscopic registration method was 0 165 sec as compared to 0 135 sec by the logarithmic method The minimum duration by the stethoscopic method was 0 105 sec, and, by the logarithmic method, it was 0 08 sec*

In every one of the 33 normal persons listed in table 8 the first heart sound was of longer duration in the stethoscopic phonocardiogram than it was in the logarithmic phonocardiogram This difference in duration is due to the greater amount of attenuation to which the first and fourth components are subjected by the logarithmic registration system Herein lies the explanation for the divergent data of table 7 [2] *In other words lack of agreement as to the duration of the first heart sound is due primarily to dissimilar frequency response characteristics inherent in the phonocardiographs employed by the investigators*

It has been shown (Fig 12) that various types of chest pieces modify the frequency response of the phonocardiograph The phonocardiograms summarized in table 8 were registered with the five centimeter diameter chest piece shown in figure 10 Such a large chest piece was employed because it

the first heart sound In our opinion such figures are misleading and have little meaning For example, it has been shown that the four elements of the first heart sound have dissimilar frequency components Under such conditions how can such a complex train of waves be expressed with any accuracy by a single frequency value? Furthermore the vibrational frequencies and the number of vibrations which comprise the first heart sound are dependent on the frequency response of the phonocardiograph and the type

TABLE 10

DURATION OF THE SECOND HEART SOUND IN 33 NORMAL PERSONS MEASURED IN STETHOSCOPIC AND LOGARITHMIC PHONOCARDIOGRAMS

Case	*Age in Years*	*Duration of Second Sound (Seconds)*	
		Stethoscopic	*Logarithmic*
1	24	0.135	0.105
2	24	0.135	0.090
3	28	0.130	0.065
4	22	0.125	0.090
5	21	0.125	0.100
6	22	0.140	0.105
7	19	0.145	0.080
8	21	0.120	0.085
9	20	0.130	0.070
10	26	0.135	0.075
11	23	0.130	0.080
12	24	0.125	0.0[illegible]0
13	22	0.120	0.050
14	22	0.110	0.085
15	21	0.130	0.110
16	22	0.100	0.060
17	22	0.100	0.070
18	21	0.105	0.090
19	21	0.095	0.0[illegible]5
20	22	0.135	0.095
21	21	0.110	0.090
22	20	0.125	0.085
23	23	0.105	0.065
24	22	0.130	0.110
25	22	0.125	0.075
26	1	0.115	0.085
27	2	0.120	0.100
28	22	0.125	0.065
29	19	0.130	0.110
30	23	0.095	0.070
31	21	0.140	0.105
32	38	0.115	0.060
33	36	0.085	0.05[illegible]

From Rappaport M B and Sprague H B

large, five centimeter bell Thus, it is obvious that more portions of the first and fourth components are lost in the phonocardiogram as the chest piece is decreased in diameter The diaphragm chest piece attenuates these heart sound components most The use of high pass electrical filters in phonocardiographic applications,[0, 1] unless adequately controlled, may also attenuate the first and fourth components of the first heart sound sufficiently to alter markedly the registered duration In ascertaining the duration of the first heart sound, the investigators listed in table 7 gave practically no consideration to these important phonocardiographic characteristics

The following are the factors, therefore, which may affect the duration of the first heart sound as sensed on the surface of the chest (1) attenuation effects by the chest structure, (2) the frequency response characteristic of the phonocardiograph, and (3) the type of chest piece employed If open bell chest pieces of small diameter are used, the application pressure is an additional modifying factor With contact or button microphones, additional factors are introduced, such as chest wall compliance and microphone stiffness The application pressure of a contact microphone affects both the chest wall compliance and the microphone stiffness, thus, making them extremely variable

A number of investigators[36, 37, 38, 40, 43, 49] have published figures on the frequency in cycles per second and the number of vibrations that compose

TABLE 9

COMPILATION OF DATA FOUND IN THE LITERATURE PERTAINING TO THE DURATION OF THE SECOND HEART SOUND IN NORMAL PERSONS

Author	*Year of Observation*	*Duration of Second Sound in Seconds*	*Method*
Einthoven W[3]	1907	0 041 0 104	Electrical
Roos E[36]	1908	0 045 0 048	Flame
Weiss O[37]	1910	0 071	Direct
Gerhartz H[38]	1911	0 07	Direct
Kahn R H[39]	1911	0 068 0 081	Electrical
Lilienstein[40]	1911	0 06	Electrical
Ohm R[41]	1912	0 052 0 061	Direct
Eyster J A E[4]	1912	0 095	Electrical
Kapff W[43]	1914	0 043 0 093	Direct
Bridgman E W[44]	1915	0 089	Electrical
Strahl E O[4]	1920	0 062 0 10	Direct
Kanner L[46]	1921	0 10	Direct
Frey W[5]	1926	0 062 0 10	Electrical
Yoshioka J[3]	1932	0 095 0 19	Electrical
Braun Menéndez E and Orias O[33]	1934	0 10 0 14	Direct

From Rappaport M B and Sprague H B

heart sound are as divergent as those published for the first heart sound

Measurements of the duration of the second heart sound, obtained on 33 normal persons, are given in table 10 These measurements were made on the same stethoscopic and logarithmic phonocardiograms from which table 8 was derived *The maximum duration of the second heart sound by the*

TABLE 12

INTERVAL BETWEEN THE BEGINNING OF THE SECOND HEART SOUND AND THE CENTER OF THE THIRD HEART SOUND IN 33 NORMAL PERSONS MEASURED IN STETHOSCOPIC AND LOGARITHMIC PHONOCARDIOGRAMS

Case	*Age in Years*	*Stethoscopic Interval Between Second and Third Sounds (Seconds)*	*Logarithmic Interval Between Second and Third Sounds (Seconds)*	*Heart Rate Per Minute*
1	24	0 185	0 175	69
2	24	0 180	0 160	78
3	28	0 185	Not Present	75
4	22	0 190	Not Present	60
5	21	0 175	0 165	75
6	22	0 205	Not Present	60
7	19	0 190	Not Present	68
8	21	0 190	Not Present	54
9	20	0 190	0 165	57
10	26	0 180	Not Present	72
11	23	0 180	0 175	66
12	24	0 160	Not Present	84
13	22	0 160	Not Present	68
14	22	0 185	0 165	63
15	21	Not Present	Not Present	66
16	22	Not Present	Not Present	81
17	22	0 160	Not Present	72
18	21	0 170	0 160	72
19	21	0 160	Not Present	66
20	22	0 200	0 185	63
21	21	Not Present	Not Present	78
22	20	0 200	Not Present	75
23	23	Not Present	Not Present	93
24	22	0 195	0 170	63
25	22	Not Present	Not Present	66
26	21	0 185	Not Present	72
27	22	0 165	Not Present	66
28	22	0 180	0 160	78
29	19	0 175	Not Present	75
30	23	0 185	Not Present	69
31	21	0 195	Not Present	57
32	38	0 240	Not Present	96
33	36	Not Present	Not Present	84

From Rappaport M B and Sprague H B [2]

of chest piece or microphone employed *To conclude, the first heart sound is a noise consisting of a conglomeration of unrelated frequencies that can not be accurately regarded as a pure harmonic vibration*

THE DURATION OF THE SECOND NORMAL HEART SOUND

Table 9 is a compilation of data by various authors on the duration of the normal second heart sound The duration measurements of the second

TABLE 11

DURATION OF THE THIRD HEART SOUND IN 33 NORMAL PERSONS, MEASURED IN STETHOSCOPIC AND LOGARITHMIC PHONOCARDIOGRAMS

Case	Age in Years	Duration of Third Sound (Seconds)	
		Stethoscopic	Logarithmic
1	24	0 085	0 035
2	24	0 080	0 020
3	28	0 050	Not Present
4	22	0 055	Not Present
5	21	0 050	0 050
6	22	0 070	Not Present
7	19	0 055	Not Present
8	21	0 050	Not Present
9	20	0 080	0 025
10	26	0 060	Not Present
11	23	0 030	0 015
12	24	0 045	Not Present
13	22	0 050	Not Present
14	22	0 065	0 015
15	21	Not Present	Not Present
16	22	Not Present	Not Present
17	22	0 060	Not Present
18	21	0 040	0 035
19	21	0 035	Not Present
20	22	0 055	0 030
21	21	Not Present	Not Present
22	20	0 040	Not Present
23	23	Not Present	Not Present
24	22	0 065	0 030
25	22	Not Present	Not Present
26	21	0 050	Not Present
27	22	0 040	Not Present
28	22	0 060	0 045
29	19	0 055	Not Present
30	23	0 060	Not Present
31	21	0 055	Not Present
32	38	0 035	Not Present
33	36	Not Present	Not Present

From Rappaport M B and Sprague H B [2]

grams showed a distinct third sound for only 10 of the normal persons (30 per cent)

Table 12 gives the intervals between the beginning of the second heart sound and the midpoint of the third sound The midpoint of the third sound was selected as a reference because it may be estimated with greater ease than the starting point *The stethoscopic method showed a maximum in*

TABLE 14

AMPLITUDE RATIO OF FIRST HEART SOUND TO SECOND HEART SOUND IN 33 NORMAL STETHOSCOPIC AND LOGARITHMIC PHONOCARDIOGRAMS

Case	*Age in Years*	*Stethoscopic Amplitude (Ratio of First Sound to Second Sound)*	*Logarithmic Amplitude (Ratio of First Sound to Second Sound)*
1	24	1 30	0 93
2	24	1 43	1 10
3	28	1 43	1 16
4	22	1 45	1 00
5	21	0 92	1 00
6	22	1 00	1 06
	19	1 20	0 6
8	21	0 71	1 00
9	20	1 04	0 92
10	26	1 27	1 27
11	23	0 8	0 84
12	24	1 00	0 68
13	22	0 85	0 5
14	22	1 44	0 92
15	21	0 83	0 70
16	22	0 90	0 62
17	22	1 00	0 5
18	21	0 94	1 11
19	21	0 66	0 75
20	22	1 00	2 00
21	21	0 93	0 7
22	20	0 67	0 59
23	23	1 00	1 13
24	22	1 11	0 88
25	22	1 07	1 00
26	21	0 91	0 91
2	22	1 04	1 43
28	22	1 18	1 19
29	19	1 20	1 20
30	23	1 00	0 54
31	21	0 2	0 73
32	38	1 00	1 09
33	36	0 71	0 64

From Rappaport M B and Sprague H B

stethoscopic registration method was 0 145 sec , as compared to 0 110 sec by the logarithmic method The minimum duration by the stethoscopic method was 0 085 sec , and, by the logarithmic method, 0 055 sec

The data in table 10 again illustrate how the first, third, and fourth components of the second heart sound are more effectively attenuated in the logarithmic than in the stethoscopic phonocardiograms in such a way as to alter the measurable duration Likewise, phonocardiographic data obtained with apparatus of dissimilar characteristics cannot be expected to correspond, as was true for the first heart sound

The second heart sound consists of a conglomeration of unrelated frequencies, as does the first heart sound Therefore, the statement which was made with regard to measurements on the frequency in cycles per second and the number of component vibrations holds just as true for the second heart sound as it does for the first

THE DURATION OF THE THIRD NORMAL HEART SOUND

The duration of the third heart sound was also measured in the 33 normal stethoscopic and logarthmic phonocardiograms (Table 11) The measurements in several of the normal graphs are approximate because of the difficulty in accurately judging the beginning and the end point of such a typically coarse vibration of low amplitude *The maximum duration by the stethoscopic method was 0 085 sec and by the logarithmic method, 0 05 sec The minimum duration by the stethoscopic method was 0 03 sec , and by the logarithmic, 0 015 sec*

The third heart sound registered clearly in 27 of the 33 stethoscopic phonocardiograms (82 per cent), whereas the logarithmic phonocardio

TABLE 13

COMPILATION OF DATA FROM THE LITERATURE PERTAINING TO THE INTERVAL BETWEEN THE SECOND AND THIRD HEART SOUNDS

Author	*Year of Observation*	*Interval Between Second and Third Sounds (Seconds)*	*Method*
Einthoven W [3]	1907	0 13	Electrical
Lewis T [5] [6]	1913	0 18	Electrical
Hess O [7]	1915	0 26	Direct
Bridgman E W [44]	1915	0 13 0 18	Electrical
Leonhardt W [31]	1932	0 115 0 15	Electrical
Clerc A Zodac Kahn B and Tavecchi G [58]	1934	0 12 0 18	Electrical
Braun Menendez E and Orias O [33]	1934	0 11 0 14	Direct
Duchosal P [59]	1935	0 11 0 18	Electrical

From Rappaport M B and Sprague H B [7]

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terval of 0 240 sec and a minimum of 0 160 sec The maximum logarithmic interval was 0 185 sec , and the minimum, 0 160 sec The difference between the stethoscopic and logarithmic measurements may be accounted for by the obliteration or attenuation of the first component of the second heart sound, which effectively shortens the interval Obviously, phonocardiographs of dissimilar frequency response will not supply similar data This is well illustrated by the data in table 13 There appears to be no correlation between heart rate and the interval between the beginning of the second heart sound and the midpoint of the third sound

Amplitude of the normal first and second heart sounds

The amplitude ratio of the first to the second heart sound in the 33 normal phonocardiograms registered at the apex is listed in Table 14[2] *In 20 of the 33 normal stethoscopic phonocardiograms (61 per cent), the amplitude of the first heart sound was greater than, or equal to, that of the second heart sound In 15 of the logarithmic phonocardiograms (45 per cent), the amplitude of the first sound was greater than, or equal to, that of the second* This difference between stethoscopic and logarithmic registration may be explained by the greater preponderance of higher frequency components in the second sound than in the first The relationship does not necessarily exist in other age groups

The normal atrial sound

We observed an atrial sound in 29 of the 33 stethoscopic phonocardiograms (88 per cent), whereas the logarithmic system registered it in only 7 of the 33 (21 per cent)[2] This illustrates that human hearing is relatively inefficient in the detection of normal atrial or fourth heart sounds because of their low frequency and low intensity

The normal systolic murmur

There was a slight trace of systolic murmur in 19 of the 33 stethoscopic phonocardiograms (58 per cent) The logarithmic registrations showed some evidence of a systolic murmur in 28 of the 33 (85 per cent) Furthermore, the logarithmic system registered the murmur whenever the stethoscopic system did[2] This indicates that the selective attenuating properties which are peculiar to human hearing tend to bring out a slight murmur more efficiently because of the greater preponderance of higher frequency components in such a murmur

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section II

heart sounds and murmurs

8 / Gallop Rhythms Triple and Quadruple Rhythms Including a Discussion of Gallop Sounds and the Splitting of the First Sound

HISTORICAL NOTES

Gallop sounds were first described by Charcellay of Tours in 1838[1], the patient was a sixty three year old man with Bright s disease At the level of the second rib an extra sound was noted which immediately preceded the first sound and the carotid pulse At postmortem examination, atrial hypertrophy was found, and Charcellay suggested that the extra sound had been atrial in origin According to Potain, Bouillaud was the first to use the term gallop rhythm " about the year 1847, but it first appeared in Potain's writings in 1875 The first phonocardiograms showing gallop rhythm were made by Weiss and Joachim[4] in 1910

DEFINITION

The term gallop rhythm' is usually applied to a triple or a quadruple rhythm occurring in a patient with heart disease who has a heart rate greater than 100 beats per minute

CLASSIFICATION

In view of the clinical importance attached to gallop rhythms, it is essential that there be some adequate classification of these sounds together with an understanding of what causes them and methods of differentiating them

Potain classified gallop rhythm depending on time relationship, as protodiastolic mesodiastolic and presystolic He believed that the first and the last types could become mesodiastolic if the heart rate became more rapid and diastole was shortened

A satisfactory classification is as follows (Fig 45)

1 Systoli gallop where the additional sound falls approximately midway between the first and second heart sound

2 Atrial (presystolic) gallop where the additional sound is due to atrial systole and precedes the first sound complex

3 Mesodiastolic gallop which occurs when an additional sound occurs in mid-diastole

Dystolic – Precedes 1st HS due to atrial contraction
Systolic – Midway between 1st & 2nd HS
diastolic Early in diastole
-diastolic In mid diastole
mation gallop – Extra sound + 3rd HS

are altered by tachycardia, but any three sounds occurring at a rapid rate have a different quality from the same three sounds at a slower rate

The significance of rate becomes more confusing when dealing with children Here a third sound is normally present and frequently the rate is well over 100 Should this be called gallop rhythm?

It would seem that any extra sound, whether presystolic, systolic, protodiastolic or mesodiastolic, occurring in a heart which is thought to be abnormal, should be labeled a "gallop," whereas the same sound in a normal heart should be given some less ominous title, such as a normal third heart sound

REFERENCE TRACINGS

Most gallop sounds represent an accentuation of normally occurring sounds which are not always audible on routine auscultation, even though they may be recorded by such special technics as esophageal recordings (in the case of atrial sounds) or low frequency stethoscopic recordings (for both atrial and third sounds) In order to study gallop sounds adequately, it is necessary to record the phonocardiogram with other reference tracings in addition to the electrocardiogram, which although adequate for examining the periods of systole and presystole, is totally inadequate for the examination of events occurring during mechanical diastole The apex cardiogram is the best reference tracing for most sounds occurring during diastole The key to the diastolic tracing of the apex cardiogram is referred to as the 0 point, this represents the beginning of the rapid ventricular filling early in diastole It is obvious that if the 0 point represents the beginning of the rapid inflow phase, then it must coincide with the opening of the atrioventricular valves, allowing the flow of blood from the atria to the ventricles The summit of the rapid inflow wave denotes the time of occurrence of the third heart sound or the protodiastolic gallop, whereas splitting of the second sound usually occurs prior to the beginning of the rapid inflow wave, and thus prior to the 0 point An exception to this may be noted when there is considerable delay in pulmonic closure, in which case the 0 point and the second part of a split second sound may coincide

Some help in timing the atrial gallop may be obtained from the jugular venous pulse tracing Orias and Braun Menendez[4] regarded the jugular pulse tracing as the best reference tracing for all gallops, but we prefer to use the apex cardiogram for early diastolic extra sounds

CHARACTER OF THE SOUNDS

The majority of gallop sounds are of low frequency and low to moderate intensity, and therefore they cannot be heard as well as the accompanying first and second heart sounds Occasionally, however, gallop sounds may be the loudest sounds in the cardiac cycle When the gallop sounds are of low

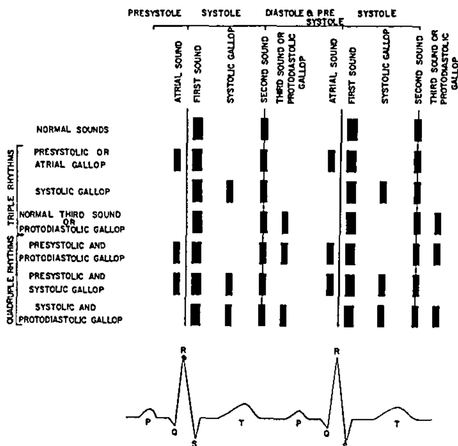


Fig 45 Schematic representation of triple and quadruple rhythms

It may be caused by atrial summation or protodiastolic gallop and is dependent on the length of diastole

4 Summation gallop which occurs at the time of the normal third heart sound in a patient in whom the atrial contraction occurs immediately prior to the expected timing of the normal third heart sound causing a summation effect This may result in the accen*tuation of a normal third sound or in the creation of an entirely new sound*

5 Protodiastolic gallop where the additional sound occurs early in diastole This is sometimes called a ventricular gallop

HEART RATE

Considerable confusion surrounds the subject of the gallop rhythm By definition gallop rhythms occur only when the heart rate is more than 100 beats per minute While it is true that the triple rhythms more closely resemble a horse's canter at faster rates, there is no good reason to suppose that a protodiastolic sound, by itself, at a rate of 110, is any more significant than the same sound at a rate of 95 The tachycardia may be significant and may affect the character of protodiastolic sound, but the sound itself is no more important at one rate than at another

Additional sounds take on different qualities at a faster rate because factors such as strength of myocardial contraction and early diastolic filling

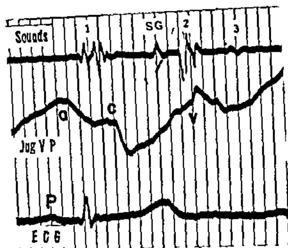


Fig 47 (Apex L g) Quadruple rhythm with lat systol c click and no mal th d heart sound pat nt h d an ppare tly normal heart Note the first sound (1) of normal ntensity commenc g 0 03 s aft r the onset of th QRS c mplex and spl t by 0 04 sec with the tw compon nts of the spl t of eq al tensity The l te systolic low f equency brat on pr sents a systol c gallop (SG) The s cond sound (2) is of normal intensity and is spl t by 0 03 sec the third sound (3) is of low int s ty and low frequency

tiation between mid systolic clicks and systolic gallops The former are said to be dry, high pitched clicking sounds and the latter are lower pitched sounds Wolferth and Margolies[6] reported 2 well documented cases, one in a 14 year old girl extremely ill with miliary tuberculosis and the other in a woman about 50 years old who had hypertension, coronary arteriosclerosis, and heart failure Mid systolic clicks are said to occur in young persons who have severe fevers or in elderly people with arteriosclerotic changes in the aorta

Systolic gallops vary from patient to patient in their relationship with the first and second heart sounds, but are usually described as occurring around mid systole The sound may be heard best close to the apex, or over the base in some cases it is affected by respiration or by the patient's position

Systolic gallops are not pathognomonic of any particular disease process

PRESYSTOLIC GALLOP

The term 'presystolic gallop' is not a good one and would be better re placed by 'atrial gallop' Presystolic gallop merely implies that an extra sound is noted immediately preceding the succeeding first sound in the cardiac cycle It is important to know whether this additional sound is due to atrial systole alone, whether it is due to early ventricular filling or to a combination of these events It is not necessary for presystolic sounds to be caused by atrial systole and one often encounters a patient with atrial

frequency and low intensity, they may be better felt with the hand than heard through the usual type of stethoscope These sounds are said by older clinicians to be best perceived by the use of a stiff wooden stethoscope, which allows one to appreciate the tactile sensation of the precordial thrust as well as actually to hear the sound vibrations themselves There are also some experienced clinicians who prefer to apply the ear directly to the chest wall for the same reasons

CLINICAL SIGNIFICANCE OF GALLOP

It is important to realize that all gallop sounds do not have the same clinical significance Atrial gallops suggest atrial hypertrophy from any cause Systolic gallops may occur in normal hearts Protodiastolic gallops are associated with myocardial embarrassment, and summation gallops are caused by atrioventricular conduction defects

SYSTOLIC GALLOP (FIGS 46 AND 47)

This often mentioned, but rarely encountered, form of triple rhythm was described first by Cuffer and Barbillion in 1887 It is seldom correctly diagnosed clinically and not often detected in phonocardiograms Most cases diagnosed clinically as systolic gallops turn out to be presystolic gallops or short systolic murmurs Their causation and clinical significance are not known

Apart from the nature of the sound, it is difficult to justify any differen

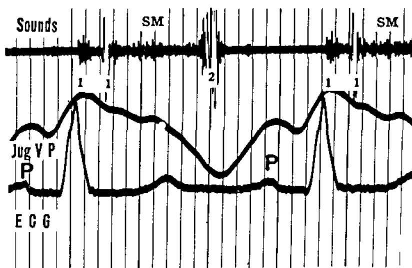


Fig 46 (2 L I S Log) Loud early systolic click in an apparently normal heart Note the two groups of vibrations labeled (1 1) which represent a low intensity first sound occurring 0 06 sec after the onset of the QRS complex followed 0 07 sec later by a narrow group of higher intensity caused by the systolic click Note also the systolic murmur (SM) of low intensity and medium frequency and the prominent second sound (2)

P wave, irrespective of whether these P waves fall during ventricular systole or ventricular diastole The atrial sounds falling during ventricular systole are presumably due principally to atrial contraction, as the atrioventricular valves are closed at this time This tracing emphasizes the fact that atrial sounds may be systolic, protodiastolic mesodiastolic or presystolic in timing It is also obvious that atrioventricular conduction defects, such as first or second degree heart block, may result in atrial sounds occurring in various parts of diastole Atrial sounds may also be encountered in atrial flutter but are not heard in atrial fibrillation

ATRIAL GALLOP

Etiology

Any disease or physiologic state causing increased left or right atrial pressure and atrial hypertrophy may produce an atrial gallop (Figs 50, 51, and 52) These conditions include (1) heart failure from any cause (such as myocarditis, coronary artery disease, hypertension from any cause aortic valve disease, cor pulmonale, severe pulmonic stenosis, severe anemia, large left to right shunts, or certain infectious fevers such as typhoid) and (2) obstructive or, less commonly regurgitant lesions involving the mitral or tricuspid valves

Detection

Atrial sounds are heard best at the apex or in the second or third interspaces, either to the left or to the right of the sternum They vary greatly

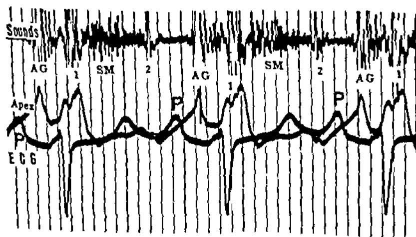


Fig 50 (Apex Log[illegible] with simultaneous electrocardiogram) Atrial gallop in severe valvular pulmonic stenosis The first sound (1) is of normal intensity and occurs 0 08 sec after the onset of the QRS The diamond shaped systolic murmur (SM) is of medium frequency and moderate intensity The second sound (2) is of normal intensity and the loud atrial gallop (AG) falls in presystole and occurs 0 14 sec after the commencement of the P wave and 0 08 sec after its peak

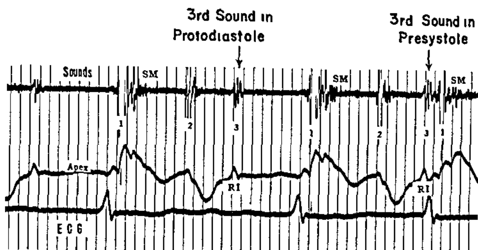


Fig 48 (Apex Log) Changing position of a gallop sound in atrial fibrillation with a third sound producing a protodiastolic gallop in one cycle and a presystolic in the next The relationship of the third sound to the preceding second sound is constant but its relationship to the succeeding first sound varies depending on the length of diastole Note the normal first (1) and second (2) sounds and the third sound (3) in protodiastole in the first cycle and in presystole in the second The third sound occurs 0 17 sec after the end of the second sound in both cycles but it precedes the succeeding first sound by 0 30 sec in the first cycle and by 0 07 sec in the second cycle

fibrillation, where, depending on the length of the cycle, a third sound, al though constant in its time relationship with the preceding second sound, will be protodiastolic in one cycle and presystolic in the next (Fig 48)

CAN ATRIAL SYSTOLE ALONE PRODUCE AN AUDIBLE SOUND?

It may be questioned by some whether atrial systole can produce not only audible sounds, but often the loudest sounds in the cardiac cycle That atrial contraction can produce sounds is shown in figure 49 This tracing of a patient with three to one heart block shows atrial sounds following each

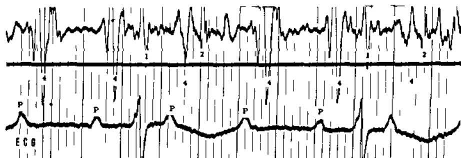


Fig 49 (Apex Steth) Atrial sounds of low frequency but high intensity occurring during ventricular systole and ventricular diastole in a patient with three to one block Note the normal first (1) and second (2) sounds and the atrial sounds (4) which follow the onset of the P waves by 0 10 sec and are of much greater intensity in ventricular diastole than in ventricular systole

or the first sound is delayed, its detection as a separate and distinct atrial sound is made easier. In contrast, when the atrial sound is of low intensity or the P R interval is shortened atrial sounds may tend to fuse with the true first sound and the detection of separate sounds is made more difficult.

Lengthening of diastole from any cause, such as slowing of the heart rate, or following a ventricular premature beat, may result in diminution of or disappearance of atrial sounds. The probable reason for this is that toward the end of a prolonged diastole, the pressure differential between the atria and the ventricles diminishes, and the effects of atrial systole are correspondingly diminished.

Duration and Frequency

Because of the low intensity of some atrial vibrations, it is difficult to measure accurately the duration of atrial sounds. The duration has varied from 0.03 to 0.12 sec., if the duration continues much longer than this, a murmur rather than a sound is heard unless the vibrations are of unusually high intensity. The frequency of atrial sounds varies between 25 and 75 cycles per second.

Timing

Electrocardiograms are the easiest and the most commonly used reference tracing in the timing of atrial gallops, but the *a* wave of the jugular venous pulse tracing or the atrial wave in the apex cardiogram may be equally useful. Atrial gallop follows the peak of the P wave of the electrocardiogram, and only those vibrations which precede the R wave of the electrocardiogram can definitely be considered atrial in origin. In patients who have short P R intervals but prolonged atrial sounds, it is not always possible to be certain where vibrations due to atrial systole cease and those due to ventricular systole commence.

MESODIASTOLIC GALLOP (FIG. 53)

The term "mesodiastolic gallop" was used by Potain simply to express the time relationship of the protodiastolic or the presystolic gallop in the presence of a rapid heart rate and hence the mesodiastolic gallop is not a specific pathologic entity. It is obvious that in the case of a protodiastolic or a presystolic gallop a relatively long diastole is necessary before it can be ascertained whether the extra sound falls just after the second heart sound or just before the succeeding first heart sound. Thus, a *shortening* of diastole can convert a protodiastolic or presystolic gallop into a *mesodiastolic gallop*. A mesodiastolic gallop may result also from a summation of effects when a protodiastolic third heart sound is reinforced by atrial contraction. This last condition occurs more frequently in the presence of a prolonged P R interval. An infrequent cause of mesodiastolic gallop may

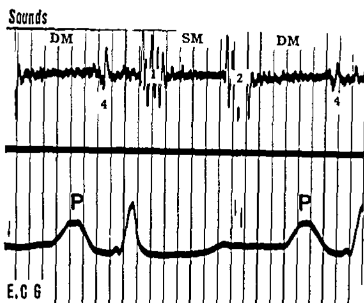


Fig 51 (2 L I S Log) Triple rhythm giving an atrial (presystolic) gallop due to an atrial sound in a patient with chronic cor pulmonale Note the first sound (1) of normal intensity occurring 0 08 sec after the onset of the QRS complex a few early systolic vibrations (SM) of low intensify and low frequency a very loud second sound (2) split by 0 03 sec with marked accentuation of the second component the diastolic murmur (DM) of low frequency *and low intensity and the prominent atrial sound (4) with the principal vibrations 0 14 sec* after the onset of the P wave of the electrocardiogram

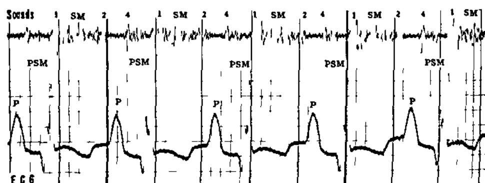


Fig 52 (2 L I S Log) Atrial sound giving an atrial (presystolic) gallop form of triple rhythm in a patient with severe pulmonic stenosis and congestive failure Note the single first sound (1) 0 07 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) of medium frequency and moderate intensity the normal second sound (2) the prominent fourth sound (4) 0 08 sec after the onset of the P wave of the electrocardiogram and *the presystolic murmur (PSM) of medium frequency and moderate intensity but without* crescendic effect

in intensity, from being virtually inaudible to being the loudest sound in the cardiac cycle Presystolic gallops due to atrial contraction are probably missed as frequently as they are recognized They are often mistaken for the first component of a split first sound or they may even be mistaken for the first sound itself when the latter is of low intensity or occurs sufficiently late to be mistaken for a systolic gallop

When the atrial sound is loud, especially if the P R interval is prolonged

of a third sound by an atrial systole or in the creation of an entirely new sound occurring at the time of a normal third heart sound and resulting from a summation of effects rather than a summation of sounds

This phenomenon usually occurs with increased heart rates and a prolonged P R interval It may occur with second degree heart block or atrial flutter, or it may be intermittent in patients with third degree atrioventricular block It does not occur with atrial fibrillation, since atrial contraction does not occur in this condition The true relationships of the occurrence of the P wave in relation to the length of diastole can easily be calculated for the production of a summation gallop

PROTODIASTOLIC OR VENTRICULAR GALLOP

Protodiastolic gallop (Fig 55) occurs at the time of a normal third heart sound It is the type of gallop rhythm most commonly encountered in adults

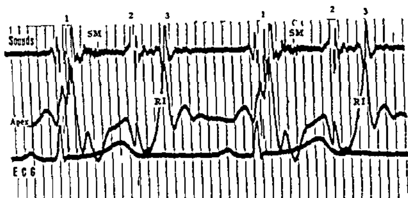


Fig 55 (Apex Log with apex cardiogram and electrocardiogram) Triple rhythm due to a loud third sound added to prominent first and second sounds Note the prominent first heart sound (1) the low intensity systolic murmur (SM) the normal second sound (2) and the prominent third sound (3) occurring at the apex of the rapid inflow wave of the apex cardiogram The heart rate in this patient was only 70 beats per minute yet the cadence of the sounds gave the effect of a gallop at a slower rate than is usually encountered

Etiology

Left or less commonly right ventricular failure from any cause may be accompanied by a protodiastolic gallop It is especially common in patients who have hypertensive heart disease or coronary heart disease, but also has been reported frequently in cases of myocarditis, severe anemia, and infectious fevers

The true cause of the extra sound is not clear, the gallop may arise from the ventricular muscle the chordae tendineae, the papillary muscles, or even the atrioventricular valves Although the cause may be variable, the timing is reasonably constant The gallop occurs at the peak of the rapid

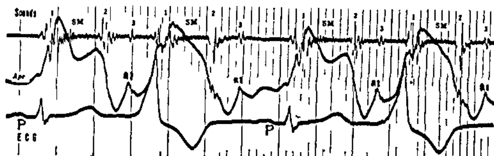


Fig 53 (Apex Log) Bigeminy with alternating protodiastolic and mesodiastolic triple rhythm resulting from ventricular ectopic beats The patient had no known heart disease and had had this arrhythmia since birth Note the relatively constant intensity of the first (1) and third (3) heart sounds The maximal vibrations at the first heart sound occur 0 08 sec after the onset at the Q wave in the normal beats and 0 13 sec after the onset at the QRS in the ventricular premature beats There is a systolic murmur (SM) of low intensity and medium *frequency slightly diminished with the ectopic beats The second sound (2) is louder following* the ectopic beat The third sound always occurs 0 16 sec after the preceding second sound and coincides with the peak of the rapid inflow wave (RI) of the apex cardiogram The third sound occurs in protodiastole following the ectopic beat and in mid diastole following the normally conducted beat

be second degree heart block with audible atrial sounds Since a normal third heart sound occurs on an average of 0 13 to 0 20 sec after the second heart sound, a diastole of approximately 0 26 to 0 40 sec in duration re sults in a mesodiastolic position of the extra diastolic sound A diastolic duration of 0 40 to 0 50 sec and a P R interval of 0 30 to 0 35 sec, with an audible sound, also results in a mesodiastolic gallop Obviously, the diagnostic significance of such a gallop sound depends not on its position in diastole, but on its underlying cause

SUMMATION GALLOP

Summation gallop (Fig 54) results from early diastolic ventricular fill ing being reinforced by atrial systole This may result in the accentuation

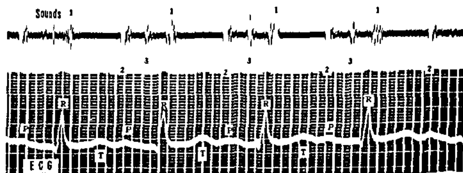


Fig 54 (Apex Log) Summation gallop in a 6 year old white boy with sinus tachycardia and first degree heart block Ventricular rate is 130 beats per minute and P R interval 0 19 sec *Note the normal intensity first sound* (1) 0 06 sec after the onset of the Q wave the normal intensity second sound (2) and the third sound (3) occurring in mid diastole 0 13 sec after the onset of the P wave and 0 11 sec after the second sound



Fig 56 (Apex L g) Quad uple rhythm due to th d and fourth sou ds in a pat ent w th c onary artery d s ase and l ft b dle bra ch block Note the no mal inte sity f st sou d (1) 0 12 sec after the onset of th QRS comple the systol c murmur (SM) of med um f que cy a d l w to mode ate i tens ty a d ar ble conf gu ation the normal ntens ty econd sou d (2) a d the lat ely lo d th d so nd (3) wh ch is the most p om nent sou d n the cycle It s p s ble in ew of the marked deg ee of bundle b a ch block that sounds 2 a d 3 r p ese t a w dely spl t second sou d Th s se ms unl kly s nce in left bundle branch block it would b ortic clos re wh ch would b delayed and e en though t might follow p lmo c cl s e it wo ld be u l k ly to do so by 0 09 sec A prom n nt atr al sound (4) occurs 0 16 sec fte the b g g of the P wa e of the elect ocard ogram

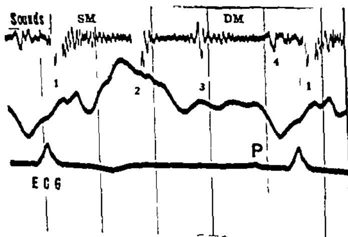


Fig 57 (Ape Log) Q d uple hythm d e to no mal f st a d second sounds an atr al sound a d a p om t th d sound n a s ely hyp rtens e yo g man who had left ent cula f l e Note the n mal inte s ty f st sound (1) 0 08 sec fte the onset of the QRS compl th d cr sc ndo sy t l mu mur (SM) of med um f equency and mode ate to l w te sity the o mal i t s ty s c nd sound (2) the prom ne t th d sound (3) 0 20 sec ft the sec d sound the m d d stol c murm (DM) of low frequ ncy nd low i tens ty a d the prom ent low f eq ency t al so d (4) app ox mately 0 12 s c afte the o set of the P w of the elect oc d og am The atr al so d and the m d diastol c murmur devel op d w th the l ft t cula d l tat on t the o set of congest e fa l r and d sappeared follow ng s pp rt th apy

inflow wave of the apex cardiogram, that is, at the time of the normal third heart sound (0 13 to 0 20 sec after the second sound)

DETECTION

Protodiastolic gallops are moderately low intensity sounds, best appreciated when the heart rate is rapid (more than 100 beats per minute in adults) and the patient is supine or in the left lateral decubitus position The sound may be intensified when the heart rate is increased by effort or excitement

CHARACTERISTICS

Protodiastolic gallops are usually considerably lower than the second *sound in intensity, but occasionally they may be higher* The frequency may be as low as 10 or as high as 40 cycles per second

EFFECTS OF ARRHYTHMIAS ON THE GALLOP SOUND (FIGS 48, 49 AND 52)

The presence of atrial fibrillation, premature beats, or atrial flutter with changing block may alter the intensity of gallop sounds and the position of the gallop, while constant in relation to the preceding second sound, may vary in relation with the succeeding first sound

RIGHT VENTRICULAR GALLOP

Gallop sounds are sometimes heard in the xiphoid region rather than at the apex In congenital heart disease these additional sounds are usually associated with left to right shunts, due either to atrial septal defects or to total anomalous pulmonary venous drainage Under these circumstances, the gallop sounds are doubtless secondary in some manner to the greatly increased blood flow from the right atrium to the right ventricle Right ventricular protodiastolic gallops may also be heard in patients who have chronic cor pulmonale, and sometimes during acute right ventricular stress following pulmonary embolism The diagnostic significance of these gallops is dependent on the underlying lesion The position of the gallop whether at the apex or at the xiphoid region is not sufficient evidence to indicate whether the gallop originates in the right or left ventricle The etiology must be known before this distinction can be made

QUADRUPLE RHYTHMS

The term 'quadruple rhythm" (Figs 56, 57 and 58) indicates the occurrence of four sounds during a cardiac cycle Any combination of four separate sounds fulfills this criterion, but the grouping most commonly encountered clinically is that of an atrial sound and a protodiastolic sound in addition to the normally occurring first and second heart sounds As can be seen from figure 58, a quadruple rhythm may also result from the addi

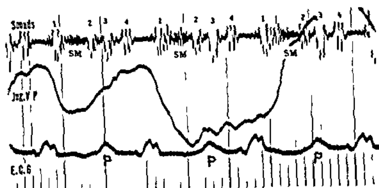


Fig 56 (Apex Log) Quadruple rhythm due to third and fourth sounds in a patient with coronary artery disease and left bundle branch block Note the normal intensity first sound (1) 0 12 sec after the onset of the QRS complex the systolic murmur (SM) of medium frequency and low to moderate intensity and variable configuration the normal intensity second sound (2) and the relatively loud third sound (3) which is the most prominent sound in the cycle It is possible in view of the marked degree of bundle branch block that sounds 2 and 3 represent a widely split second sound This seems unlikely since in left bundle branch block it would be aortic closure which would be delayed and even though it might follow pulmonic closure it would be unlikely to do so by 0 09 sec A prominent atrial sound (4) occurs 0 16 sec after the beginning of the P wave of the electrocardiogram

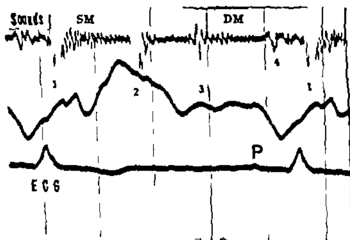


Fig 57 (Apex Log) Quadruple rhythm due to normal first and second sounds an atrial sound and a prominent third sound *in a severely hypertensive* young man who had left ventricular failure Note the normal intensity first sound (1) 0 08 sec after the onset of the QRS complex the decrescendo systolic murmur (SM) of medium frequency and moderate to low intensity the normal intensity second sound (2) the prominent third sound (3) 0 20 sec after the second sound the mid diastolic murmur (DM) of low frequency and low intensity and the prominent low frequency atrial sound (4) approximately 0 12 sec after the onset of the P wave of the electrocardiogram The atrial sound and the mid diastolic murmur developed with the left ventricular dilatation at the onset of congestive failure and disappeared following supportive therapy

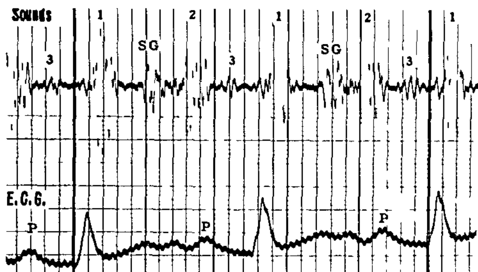


Fig 58 (Apex Log) Quadruple rhythm resulting from normal first and second sounds a *systolic gallop and a normal third sound in a young girl* who had a history of attacks which suggested parosyxmal tachycardia and in whom catheterization revealed the right side of the heart to be normal Note the prominent first sound (1) 0 06 sec after the onset of the QRS complex the mid systolic gallop (SG) the normal second sound (2) and the normal third sound (3)

tion of a systolic gallop and a third sound, together with normal first and second sounds

In some cases of bundle branch block, wide splitting of the first and second sounds, especially with tachycardia, may result in a quadruple rhythm, similarly, wide splitting of either the first or the second sounds, together with an atrial sound or a third sound, has a similar effect

Other quadruple rhythms occur in mitral stenosis with a clearly audible opening snap, together with a third sound or an atrial sound Thus, it is obvious that there may be many combinations of sounds resulting in quad ruple rhythms, and frequently a phonocardiogram, together with a selection of reference tracings, is necessary for the correct interpretation of these sounds

Split first heart sound

Split first sounds occur frequently in normal persons but also in cases of bundle branch block *They are characterized as follows*

1 Both components are of about equal intensity although either one may be louder than the other The sounds may be normal diminished or accentuated in intensity

2 The sounds are heard best at the apex or between the apex and the lower left sternal border

3 Usually, each component is of short duration

4 Each component occurs after the Q *wave of the electrocardiogram*

5 The duration of the splitting may be up to 0 06 sec

6 The sounds do not precede the apical thrust

It is generally accepted that a split first heart sound is due to asynchronism in contraction of the right and left ventricles, but it could be due to a delayed opening of the semilunar valves, (for example, the third component of the first sound) although this is much less likely

The frequency of splitting of the first heart sound in intraventricular conduction defects supports the first theory, and its presence, even with normal conduction does not contradict this, as mechanical events are not necessarily directly related to electrical events Katz,[7] in 1925, showed that there could be considerable asynchronism in the beginning of ejection of blood from the two normal ventricles

EARLY SYSTOLIC CLICKS (FIG 59)

The differentiation of the early systolic sound associated with dilatation of the pulmonary artery or of the aorta from the split first heart sound may be difficult it may necessitate the taking of simultaneous phonocardiograms and either pulmonary artery or carotid artery pressure tracings If this is done it will be seen that the two components of the split first heart sound occur before the rise in pressure in the artery concerned whereas the early systolic sounds associated with lesions of the pulmonary artery or aorta are not recorded until after the pressure rise has commenced The *differentiation* of both of these groups of sounds from the atrial gallop can be made easily by correlation with the electrocardiogram The atrial gallop must

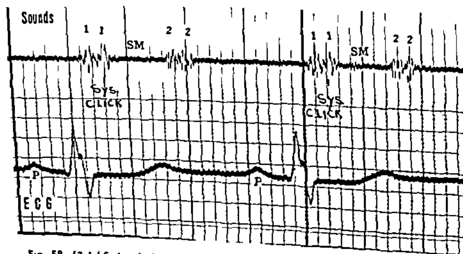


Fig 59 (2 L I S L g) Q ad ple hythm r sult g fr m a no mal f rst sound an early systol cl k nd a well spl t second sou d in a pat ent w th an atrial septal defect and pulm a y sc le bst uct on Note the normal ntens ty f rst heart so nd (1) 0 10 sec afte th o et of the QRS complex a e ly systol c cl ck (labeled l abo and sys cl ck below) o u g 0 16 s c ft r the o s t of the QRS complex and sound ng cl n cally l ke the s co d c mpone t of w dely spl t f st heart so nd the minimal systol c murmur (SM) a d th w ll spl t (0 05 s c) s cond sou d (2 2)

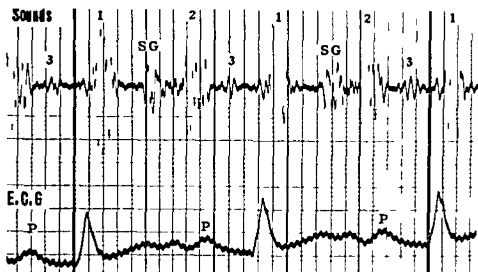


Fig 58 (Apex Log) Quadruple rhythm resulting from normal first and second sounds a systolic gallop and a normal third sound in a young girl who had a history of attacks which suggested parosyxmal tachycardia and in whom catheterization revealed the right side of the heart to be normal Note the prominent first sound (1) 0 06 sec after the onset of the QRS complex the mid systolic gallop (SG) the normal second sound (2) and the normal third sound (3)

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3 Usually, each component is of short duration

4 Each component occurs after the Q wave of the electrocardiogram

5 The duration of the splitting may be up to 0 06 sec

6 The sounds do not precede the apical thrust

9 / Splitting of Heart Sounds

FIRST HEART SOUND

Splitting of the first heart sound is encountered often in healthy persons (Fig 60) This splitting is heard best at the mitral and tricuspid areas and has been assumed to be due to asynchronous closure of the mitral and tricuspid valves, with the former closing first This splitting is heard best during moderate inspiration The interval between the two major components of the first sound rarely exceeds 0 02 to 0 03 sec

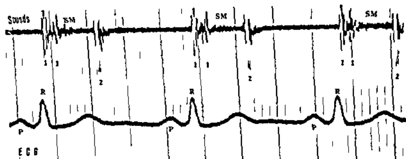


Fig 60 (2 L I S Log) Early systolic click in an apparently healthy man Note the normal intensity first sound (1) 0 08 sec after the onset of the QRS complex and the prominent early systolic click also labeled (1) as the second part of what was thought clinically to be a widely split first sound The click occurs 0 15 sec after the onset of the QRS complex Note also the low intensity systolic murmur (SM) and the normal second sound (2)

In order to prove the origin of these two components of the first sound it would be necessary to perform catheterization of both the left and right sides of the heart and to show that the first component is almost synchronous with the onset of the rise in pressure in the left ventricle and that the second component is almost synchronous with the onset of the rise in pressure in the right ventricle It would also be desirable to time the openings of the aortic and pulmonic valves in order to be certain that they are not the cause of the second part of the split It has been stated that since the second valvular component of the first sound occurs during the upstroke in the carotid artery this sound must be caused by closure of the tricuspid valve Of course this is not necessarily true, it simply proves that this component is due neither to mitral closure nor to aortic valve opening, but it

occur after the P wave and before the Q wave, whereas both of the previous groups must appear after the Q wave

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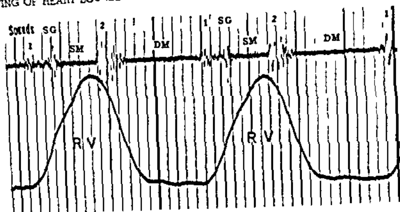


Fig 61 (2 L.I S Log and simultaneous right ventricular pressure curve) Early systolic click (SG) in an asymptomatic patient with idiopathic dilatation of the pulmonary artery Note the moderately low intensity first sound (1) almost simultaneous with the onset of pressure rise in the right ventricular cavity the early systolic click (SG—systolic gallop) 0 08 sec after the first sound a few medium frequency low intensity systolic vibrations (SM) a prominent second sound (2) and a few very low frequency diastolic vibrations labeled DM but probably representing artifacts on the baseline

The true cause of these early systolic clicks is not obvious but it is thought that the sounds occur in the aorta or the pulmonary artery It has been suggested that dilated vessels "bring ejection vibrations which are normally inaudible closer to the chest wall"[1] This may be part of the explanation but obviously it is not the whole story or we would hear this sound more frequently in persons with pectus excavatum It seems more likely that the altered dynamics either result in a marked accentuation of normal vibrations or create an entirely new set of vibrations

Another factor which has not been clearly defined in cases of valvular pulmonic and aortic stenosis is the real possibility that a loud sound may be created by sudden tensing of the appropriate semilunar valve during isometric contraction These early systolic ejection sounds occur 0 04 to 0 14 sec after the closure of the mitral valve Occasionally, patients with right bundle branch block show wide splitting of the first sound and it is believed that this is due to the late onset of right ventricular systole and thus to the late closure of the tricuspid valve

SECOND HEART SOUND

Careful auscultation of the second heart sound may be one of the most rewarding features of an entire cardiac examination. The presence or absence of splitting the duration of splitting and the actual and relative intensities of the aortic and pulmonic components may all provide valuable clues in cardiac diagnosis

It is well known that the second sound is split at the pulmonic area under normal circumstances and that the duration of this splitting increases with

does not prove whether it is due to tricuspid closure, pulmonic valve opening, or to the generation of a sound in the aorta or the pulmonary artery Since it is not possible to measure right and left ventricular pressure curves and aortic and pulmonary artery pressure curves in all cases, we shall assume that the usual cause of the split first sound is asynchronous closure of the mitral and the tricuspid valves

A split first sound must be differentiated from an atrial sound plus a first sound, and from a first sound plus an early systolic ejection sound

Although these distinctions are usually made without too much difficulty on auscultation, nevertheless, there are times when the electrocardiograph is needed to time the atrial sound, and aortic or pulmonary artery pressure curves are necessary to time the early ejection sounds

Usually, atrial sounds are of lower intensity and lower frequency than are the valvular components of a normal first sound, and they are frequently separated from the first sound by a much greater interval than the 0 02 to 0 03 sec which is the average interval between the components of a split sound Occasionally the atrial sound may be loud and may occur close to the first sound, making identification difficult The problem may be solved by the electrocardiogram, as any sound occurring before the onset of the QRS complex of the electrocardiogram cannot be part of the first sound, and if a P wave is present with a normal ventricular rate, it may be assumed that the sound following the P wave and preceding the QRS is atrial in origin Difficulties may arise in a patient who has a short P R interval, where the atrial sound may actually follow the onset of the Q wave on the electrocardiogram, or in a patient who has a rapid heart rate and a short diastole, where a third sound may follow the P wave on the electrocardiogram

The differentiation of a split first sound from an early systolic click (Fig 61) may be difficult In a patient who has a dilated aorta due to aortic stenosis, severe aortic insufficiency, hypertension from any cause, aneurysm of the aorta, aortic arteriosclerosis or dilatation of the pulmonary artery due to a mild to moderate degree of pulmonic stenosis, idiopathic pulmonary artery dilatation, or pulmonary hypertension secondary to mitral stenosis or secondary to left to right shunts, it may be assumed that an additional sound in early systole is an ejection sound This sound occurs during the rise in pressure in the aorta or the pulmonary artery, depending on the underlying lesion Usually, the additional sound in these patients is high pitched, and it may be moderate to high in intensity It has a sharp clicking quality and is heard best over the aortic and pulmonic areas, it may also be heard well at the apex This sound may be the most prominent sound in the cardiac cycle, and if the first sound is diminished, the two sounds may be mistaken for an atrial sound plus a loud first sound This early ejection sound is heard best during forced expiration, is usually considerably diminished when the breath is held in deep inspiration, and is constant in its position in the cardiac cycle

during inspiration in the normal person, it is best to assess abnormal splitting during normal expiration when splitting is minimal (Fig 62)

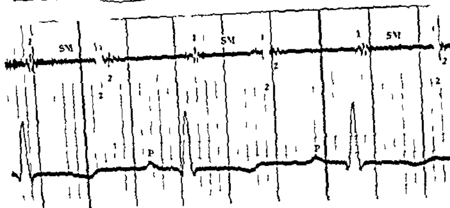


Fig 62 (2 R I S Log) The effect of resp a on on sp tt g of the second heart sound recorded at the aortic area Th s trac ng shows a change f om moderate insp at on moderate e p rat on in a p t ent w th coa ct t on of the ao ta Note the F st sound (1) c s st ng of two g o ps of low ntens ty b at ons spl t by 0 03 s Th s sou d a d ts spl tt are constant n each cycle In the f st r orded seco d s u d (2) there are two ob component the first be ng m ch loude th the s cond The du at on of spl tt ng s 0 04 As the pati nt b eathes out the second component of the second sound (the pulmon c closure) comes close a d closer to the first component (the aort c component) unt l t sounds f se

It should be remembered that pulmonic closure is not always loudest the second left interspace and is frequently *heard* best at the third, occasionally in the first left interspace It should also be realized tha congenital heart disease with various anomalies of the great vessels, sound heard in the second left interspace may not be due to pulmonic v closure

The importance of a split second sound is that it implies the presenc sounds from two semilunar valves, an aortic and a pulmonic On the c hand absence of splitting *does* not necessarily *mean that* only one val present it means simply that the closure of only one valve is audible conceivably that the valves close simultaneously For example, in pat who have clas ical tetralogy of Fallot with infundibular pulmonic ster the second sound is single and is heard best at the third to fourth left space This sound is thought to be *due to* aortic valve closure, as pulr closure which occurs almost simultaneously under these circumstanc greatly reduced in intensity and is *inaudible* as a separate sound

Wide splitting of the second sound may be due to prolongation of ventricular systole (in which case the normal relationship of aortic pulmonic valve closure is maintained in that aortic valve closure c first) or it may be due to a considerable prolongation *of left* ventr systole (in which case pulmonic valve closure occurs first) A third br

inspiration. The generally accepted reason for this is the fact that the two audible components of the split are the closing of the aortic and pulmonic semilunar valves and that normally the aortic closure occurs first. Increased right ventricular flow with inspiration results in delayed closure of the pulmonic valves and accounts for the increased duration of the splitting.

The relative intensities of the aortic and pulmonic components of the second sound vary with different ages in normal persons and vary also in accordance with different pathologic states.

In normal infants and children pulmonic valve closure is usually more noticeable than aortic valve closure, and *the reverse is true in normal adults.* Some of this change may be due to the nearness of the pulmonic valve to the chest wall in children and to the gradually increasing aortic pressure *in adults. The often recorded statements that P2 is greater than A2 or vice versa* are meaningless unless it is stated that P2 is greater than A2 because P2 is greater than normal or because A2 is less intense than normal, or that *A2 is greater than P2 because P2 is less intense than normal or A2 is of* greater intensity than normal.

SPLITTING OF THE SECOND HEART SOUND

It is important to appreciate the minimal degree of separation of the two valvular components of the second heart sound which can be detected by the physician. It is not at all difficult for the average medical student to detect splitting of 0.05 to 0.06 sec., and with training and experience one can confidently detect splitting of 0.03 sec. It must be appreciated that these statements refer to the complex noises known as heart sounds and not to pure tones which may be detected at much narrower intervals.

Various factors affect the ability of the human hearing mechanism to detect the splitting of heart sounds. If the first component of a narrow splitting is unusually loud, fatiguing of the ear results, and the second component may be inaudible. This situation becomes more marked if the second component is diminished in intensity.

Splitting of the second sound is heard best at the second left interspace and along the left sternal border. In adults the first part of the splitting is usually identical in timing with the single component of the second sound as heard at the aortic area and at the apex. This is not quite the same in children, in whom splitting of the second sound is often heard clearly at the aortic area, and in whom a single second sound, if heard at the apex, may be due to the more prominent pulmonic valve closure. A simultaneous aortic or carotid artery tracing can be used to time aortic valve closure by using the dicrotic notch.

With normal breathing the interval between aortic and pulmonic valve closure during expiration averages 0.02 to 0.03 sec.; on inspiration it increases to from 0.04 to 0.05 sec. Deep inspiration *may increase the splitting* to 0.08 or rarely to 0.10 sec. Because of the extreme variability of splitting

Paradoxical splitting of the second sound may be difficult to elicit in the presence of aortic stenosis, as pulmonic valve closure may be obscured by the aortic systolic murmur. This sign is more easily appreciated in the presence of left bundle branch block.

INCREASED DURATION OF SPLITTING SECONDARY TO PULMONARY DISEASE. Since normal physiologic splitting of the second sound at the pulmonic area increases during inspiration, and since this increase is due to delayed closure of the pulmonic valve following increased venous return and increased output from the right side of the heart, it follows that increased intrathoracic negative pressure will accentuate this course of events. This can be seen easily with deep inspiration, but it occurs also with obstruction to the inspiratory pathway and in the presence of lesions preventing expansion of the lungs themselves but not affecting the thoracic cage (for example, fibrosis affecting the lungs or the pleura).

EFFECTS OF LOUD PULMONIC VALVE CLOSURE. In pulmonary artery hypertension caused principally by increased flow, the second sound at the pulmonic area is usually split and the second component is accentuated. If, on the other hand the increased pressure is due to increased pulmonary vascular resistance, there may be tremendous accentuation of the pulmonic second sound, and the sound may appear single.

In the presence of increased pulmonary flow, occasionally the second sound is single on auscultation, as aortic valve closure may be masked by the shunt murmur, but pulmonic closure is more or less delayed because of the increased duration of right ventricular systole. In the pulmonary vascular obstruction syndrome, aortic valve closure may be masked by the almost simultaneously occurring, greatly accentuated pulmonic valve closure. Under these circumstances, splitting may be elicited better at the aortic area or even at the mitral area.

Although accentuation of the pulmonary second sound is our best clinical guide to increased pulmonary artery pressure and although an accentuated aortic second sound suggests increased aortic pressure, it is important to emphasize that an accentuated pulmonic or aortic second sound does not necessarily prove the presence of pulmonary artery or systemic hypertension. The excitability of a patient, the thinness of the chest wall, and the presence of dilatation of the pulmonary artery or the aorta may all serve to bring about apparent accentuation of semilunar valve closure at the pulmonic or aortic areas.

It is more important to detect selective accentuation or diminution of a particular sound relative to the other sounds in the cardiac cycle than to record simply that a particular sound is of greater or lesser intensity than normal.

DIMINISHED AORTIC OR PULMONIC VALVE CLOSURE SOUNDS. A diminished pulmonic or aortic second sound does not necessarily mean that disease affects the valve; in fact, it does not even necessarily imply heart disease.

striking cause is shortening of left ventricular systole, as may occur in mitral insufficiency

DELAY IN PULMONIC VALVE CLOSURE. Pulmonic valve closure may be delayed under three main sets of circumstances

1 Late activation of the right ventricular musculature in some cases of right bundle branch block This is not a common cause

2 Increased right ventricular systolic output as in large left to right shunts due to anomalous pulmonary venous drainage, atrial septal defects and sometimes to ventricular septal defects Large atrial defects with low pulmonary vascular resistance and large pulmonary flow are the best examples of this group

3 Prolonged right ventricular systole in the presence of severe pulmonic stenosis with an intact ventricular septum

The duration of splitting of the second sound in pulmonic stenosis with an intact ventricular septum is a manifestation of the severity of the stenosis and of the functional capacity of the right ventricle Splitting of the second sound under these circumstances is easily missed The aortic valve closure may not be audible at the pulmonic area because of the loud systolic murmur which often envelops the aortic valve closure, and, in the more severe cases, the greatly diminished pulmonic valve closure may not be audible at the aortic area Careful listening is required for identification of the aortic closure at the second right interspace, then further careful listening may be required to detect the diminished and delayed pulmonic valve closure at the second left interspace

DELAY IN AORTIC VALVE CLOSURE Depending on the degree of delay in aortic valve closure, splitting of the second sound may be lessened, normal, or increased, in accordance with the relationship of aortic and pulmonic closure With a slight delay of aortic closure, the second sound will be narrowly split or may, in fact, be single If aortic closure becomes sufficiently delayed to follow pulmonic closure, there may be more or less splitting of the second sound, depending entirely on the interval by which aortic closure follows pulmonic It is under these circumstances that one encounters paradoxical splitting of the second sound This simply means that during inspiration the two components of the second sound occur closer together rather than further apart The obvious reason for this is that with the normal delay of pulmonic closure during inspiration, pulmonic closure more and more approaches the timing of the delayed aortic valve closure Under these circumstances a second sound which appears single with quiet breathing *may become definitely split on deep expiration*

Delayed aortic valve closure occurs under two main sets of circumstances

1 Delayed onset of left ventricular contraction as in marked left bundle branch block

2 True prolongation of left ventricular systole as may occur in severe aortic stenosis or patent ductus arteriosus

and ectopic premature beats, all may lead to diminished intensity of aortic valve closure

SPLITTING OF THE HEART SOUNDS IN RIGHT BUNDLE BRANCH BLOCK

Splitting of the first heart sound in right bundle branch block occurs more commonly than abnormal splitting of the second sound, but, even so, it does not occur every time as one might expect The reason for the lack of splitting is not apparent By contrast, in many patients who have ventricular premature beats there is marked splitting

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Emphysema, if severe, may cause the heart to be covered with lung tissue, in which case semilunar valve closure may be almost inaudible Obesity and anasarca also create severe difficulties in auscultation Myocardial lesions not specifically affecting the valves themselves, and such diseases as myxedema, amyloid disease, various forms of myocarditis, or pericarditis, constrictive or with effusion, may all result in diminution of the intensity of the heart sounds

These and many other factors have to be considered in the interpretation of the intensity of the various sounds encountered during auscultation In spite of careful consideration of all obvious modifying factors, occasionally a patient is encountered who has valid pulmonary artery hypertension and an apparently normal pulmonic valve closure, conversely, a patient may have a strikingly loud pulmonic valve closure and normal pulmonary artery pressure

These apparent contradictions in no way lessen the value of auscultation, but simply help to prevent one from becoming too dogmatic

SINGLE SECOND SOUNDS

MASKING EFFECT OF MURMURS In certain pathologic states the second sound may appear single because of the listener's inability to hear an aortic sound which, nevertheless, may be of normal intensity In severe mitral incompetence and in ventricular septal defects, systolic murmurs may occur up to or slightly beyond aortic valve closure, as the resistance of flow into the left atrium or the right ventricle may be less than that into the aorta The aortic valve closes when left ventricular pressure falls below aortic pressure, but this does not mean that the flow from the left to the right ventricle, or from the left ventricle to the left atrium, will stop precisely at this time It is true that a rapid drop in left ventricular pressure results in all of these events occurring in quick succession, but it is conceivable that aortic valve closure may occur first and may be obscured by the murmur of a ventricular defect or of mitral insufficiency A similar obscuring of the aortic second sound occurs in valvular pulmonic stenosis In patent ductus arteriosus or in aortic pulmonary fenestration, where the maximum blood flow from the aorta to the pulmonary artery occurs about the time of aortic closure, it is common for the continuous machinery murmur to obscure aortic and also pulmonic valve closure

SINGLE SECOND SOUND DUE TO DIMINISHED INTENSITY OF AORTIC VALVE CLOSURE Some patients with severe aortic stenosis have a definite diminution in the intensity of aortic valve closure, probably due to the lack of mobility of the scarred, fixed cusps This sound may also be diminished when there are lesions causing severe aortic insufficiency (rheumatic aortic insufficiency, perforation of valve cusps with bacterial endocarditis, or rupture of a sinus of Valsalva into the left ventricle) Poor muscle tone in myocarditis, low aortic diastolic pressure with shock or severe tachycardia,

the early stages of development, and the mere desire to collect data from patients with supposedly "innocent," or "functional," murmurs is not an indication for this form of investigation

EXTRACARDIAC MURMURS

Often it is impossible to decide whether a murmur is intracardiac or extracardiac in origin, and since the terms "cardiorespiratory" and "pleuropericardial" frequently are used with little real meaning and still less proof, it might be better to admit our inability to classify these murmurs accurately from an etiologic point of view, and to use some other term, such as "not significant" to imply that the murmur is not associated with significant heart disease

Frequently it is not possible to decide after one examination whether a murmur is significant or not The past history, the presence or absence of abnormalities revealed by x rays or electrocardiograms, and follow up examinations may be required before a final diagnosis can be made

Cardiorespiratory murmurs are said to be impossible to distinguish from mitral regurgitant murmurs at times but the occurrence of a cardiorespiratory murmur mimicking both the intensity and the conduction characteristics of the mitral regurgitant murmur is likely to be extremely rare The explanation that cardiorespiratory murmurs are due to compression of the lung during diastole and to the release of this compression with the sudden entry of air into the lung during cardiac systole is difficult to accept. These (1) murmurs vary considerably with respiration (2) sounding more superficial in the chest than do the mitral regurgitant murmurs, they often (3) disappear with changes in the patient's position Frequently (4) they occur during the latter part of systole (5) They may also disappear completely during some part of the respiratory cycle, not necessarily at full inspiration

APICAL SYSTOLIC MURMURS IN DIAGNOSIS OF MITRAL INSUFFICIENCY

The causes of apical systolic murmurs of mitral insufficiency are many (see mitral insufficiency p 171) It should be stressed that there is nothing in the quality intensity or conduction of the apical systolic murmur which aids in the differentiation of the underlying cause or causes

In attempting to correlate the loudness of the mitral systolic murmur with the degree of associated mitral insufficiency there are several factors to be considered These include the size and shape of the mitral valve orifice, the velocity of the blood flow in the regurgitant jet, the viscosity of the blood and the degree to which the mitral valves and the attached chordae tendineae and papillary muscles are able to vibrate These variables cannot be assessed accurately by the use of the stethoscope alone but appreciation of them enables us to understand that some patients who have free mitral regurgitation with a large mitral orifice may have little or no audible apical

10 / Apical Systolic Murmurs

Apical systolic murmurs have a variety of underlying causes, but those occurring with significant heart disease usually indicate mitral insufficiency.

In the early part of the twentieth century, these murmurs were regarded as having a bad prognosis, then as the results of long term follow up examinations became known, especially those of World War I soldiers, the pendulum swung the other way, and in 1926 Cabot[1] stated, "Systolic murmurs without other signs of cardiac disease are of no importance as evidence of valve lesions," and that, "A diagnosis of mitral regurgitation without stenosis is never justified." As usual, the truth seems to lie somewhere between the two extremes, and, today, most physicians accept both concepts of mitral insufficiency—that of an isolated lesion as well as that of mitral insufficiency associated with mitral stenosis.

The unreliability of fluoroscopy, electrocardiography, and electrokymography in the evaluation of apical systolic murmurs would leave the observer on dangerous grounds were it not for the exploring finger of the surgeon. During recent years, the frequency with which surgeons have detected significant regurgitant jets of mitral insufficiency at operation when the condition had not been suspected clinically, has been a source of considerable embarrassment to the physician.

DIFFERENTIAL DIAGNOSIS

All physicians encounter apical systolic murmurs in patients in whom roentgenologic and electrocardiographic examinations reveal no other signs of heart disease, and whose past history and lack of symptoms give no reason to suspect the presence of any form of heart disease. At postmortem examination, some of these persons have been found to have anatomically normal hearts. Of course, this does not rule out the possibility of a functionally abnormal heart during life, at the present time, there seems to be little likelihood of this problem being solved. The supposed rarity of organic pulmonic stenosis in the past, and its relative frequency since the introduction of cardiac catheterization, should warn us to be wary of drawing final physiologic conclusions from gross anatomic appearances. The problem is more difficult in the consideration of lesions of the left side of the heart, such as mitral regurgitation. Catheterization of the left side of the heart is still in

11 / Apical Diastolic and Presystolic Murmurs Due to Causes Other than Mitral Stenosis

Apical diastolic or presystolic murmurs always indicate structural or hemodynamic abnormalities of the heart and must not be considered as innocent murmurs. Although they are most frequently caused by mitral stenosis, nevertheless there are many other causes of these murmurs or of auscultatory phenomena which may simulate them.

In this chapter we shall discuss why many lesions may be accompanied by apical diastolic murmurs and will mention briefly certain anomalies which are not presented more fully elsewhere in the book. Most of these conditions can be easily distinguished from mitral stenosis by their history, the age of discovery of the murmur, or various other characteristic clinical, electrocardiographic, or roentgenologic observations. We shall not include murmurs which are tricuspid in origin and which transmit across to the cardiac apex, such as the diastolic rumble of atrial septal defect.

CHARACTERISTICS

These murmurs are of low to moderate intensity and are usually of low or occasionally medium frequency. They commence at the time of the normal third sound and continue through mid diastole or even into presystole. Usually they do not terminate with a presystolic crescendo. Because of this great variability in intensity and duration, some murmurs are heard over a large area surrounding the apex while others may be confined to an area no more than 1 or 2 inches in diameter. Like the murmur of mitral stenosis these murmurs are heard best when the patient is in the left lateral decubitus position with the breath held in moderate expiration.

CAUSES

In general apical mid diastolic murmurs are associated with one or more of three main structural or physiologic abnormalities:

1. Increased flow across a normal mitral valve
2. Flow across a normal mitral valve into a dilated left ventricle
3. Obstruction of the mitral orifice

systolic murmur, whereas others with tight mitral stenosis and little regurgitation may have loud systolic murmurs

APICAL SYSTOLIC MURMURS IN CONGENITAL HEART DISEASE

Whereas apical diastolic murmurs are common in congenital heart disease, apical systolic murmurs are relatively rare Congestive failure, secondary to some left sided lesion such as coarctation of the aorta, severe aortic stenosis, endocardial fibro elastosis, or myocarditis, may be accompanied by mitral regurgitation as part of the picture of general cardiac dilatation or failure The mitral valve itself may be deformed by endocardial fibro elastosis, with mitral insufficiency resulting Congenital defects involving the mitral valve are not common, but in atrioventricularis communis or in the septum primum type of atrial septal defect, with corrected transportation, and occasionally with ventricular defects or secundum atrial defects, the mitral valve may be structurally malformed, causing mitral insufficiency *accompanied by the typical blowing apical systolic murmur*

APICAL SYSTOLIC MURMURS OF UNKNOWN ORIGIN

A patient may have a loud apical systolic murmur for which there appears to be no adequate explanation Sometimes these murmurs are affected by position or respiration, or they tend to come and go with no apparent reason, often they have a peculiar quality and may sound quite unlike the usual cardiac murmurs In some of these instances postmortem examination has revealed no apparent cardiac lesion

SUMMARY

A loud apical systolic murmur is almost always clinically important It *is possible that the underlying cause* (such as acute cardiac dilatation) may be only temporary and may respond to some form of treatment Nevertheless, the murmur at the time of auscultation must be considered as indicating significant heart disease

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complete atrioventricular block, in both of which a large volume of blood flows into the ventricles early in diastole The one factor common to all these lesions is increased flow across a normal, or perhaps a dilated, mitral valve, this results in relative mitral stenosis, with the production of a mur mur

APICAL DIASTOLIC MURMURS WITH RUPTURE OF THE INTERVENTRICULAR SEPTUM FOLLOWING MYOCARDIAL INFARCTION Rupture of the interventric ular septum may result in the classical lower left sternal border systolic murmur of ventricular septal defect together with an apical diastolic rumble This lesion should be suspected if these murmurs appear suddenly in a pa tient following an anteroseptal or posteroseptal myocardial infarction

FLOW ACROSS A NORMAL MITRAL VALVE INTO A DILATED LEFT VENTRICLE (FIGS 65, 66, AND 67)

In 1923 Wood and White[1] reviewed a series of patients who had apical diastolic murmurs but no mitral stenosis The one factor which appeared common to these lesions was left ventricular dilatation, and, although this is undoubtedly important in many of the anomalies which cause increased flow across the mitral valve, it appears to be the principal factor in the fol lowing conditions

1 Various forms of myocarditis (*acute rheumatic myocarditis*, infectious myocarditis and endocardial fibro-elastosis without valvular *involvement*)

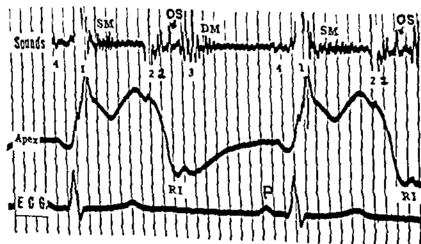


Fig 65 (Apex Log and I nea) Ap cal m d d astol c murmur w th an opening sound and a third sound in a 9 year old patient w th progeria and left ventricular d latat on following myoca d al nfa ct on Note the normal f st sound (1) 0 06 s c after the onset of the QRS comple the low intensity decrescendo systol c murmur (SM) the p om nent second sound (2 2) spl t by 0 03 sec w th the first component much louder than the second the loud th d h art sound (3) the m d d astol c mu mur (DM) following the th d heart sound the low f eq ncy low intens ty at al sound (4) and well r co ded between the s cond and th rd heart sounds The opening of the atrioventr cular val es is 0 10 sec after the beg nning of th second heart sound

INCREASED FLOW ACROSS A NORMAL MITRAL VALVE (FIGS 63 AND 64)

The most outstanding causes of apical mid diastolic murmurs in this group are the large left to right shunts, either at the ventricular level through a ventricular septal defect or at the aortic level via a patent ductus arteriosus, an aortic pulmonary artery fistula, or a ruptured sinus of Valsalva. Less common causes are peripheral arteriovenous fistulas, either congenital or acquired, and other lesions causing increased cardiac output, such as severe anemia or, rarely, thyrotoxicosis. Perhaps we should also include here the apical diastolic murmur associated with mitral insufficiency and that of

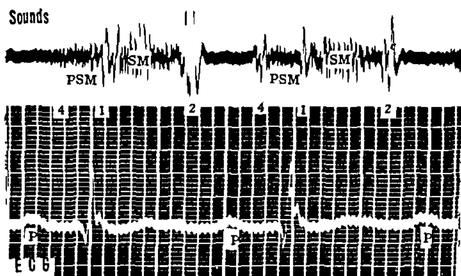


Fig 63 (Apex Log.) Presystolic murmur associated with a septum primum type of atrial defect. Note the normal first heart sound (1) 0.06 sec. after the onset of the QRS complex, the systolic murmur (SM) of moderately high intensity and medium frequency, the moderately loud second heart sound (2) split by 0.02 sec., the atrial sound (4), and the presystolic murmur (PSM) not crescendic following the fourth sound.

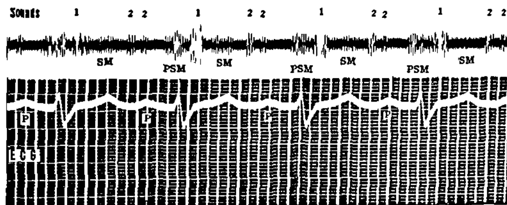


Fig 64 (Apex Log.) Apical systolic and presystolic murmurs associated with a cleft medial cusp of the mitral valve in a septum primum defect. Note the normal intensity first sound (1) 0.09 sec. after the onset of the QRS complex, the holosystolic murmur (SM) of medium frequency and low to moderate intensity, the widely split (0.06 sec.) second heart sound (2 2), and the crescendo decrescendo presystolic murmur (PSM).

tolic or presystolic murmurs in persons with normal mitral valves as shown at postmortem examination Seventeen had aortic regurgitation and the other 29 had marked cardiomegaly from various causes

AUSTIN FLINT MURMUR The Austin Flint Murmur[4] is a presystolic murmur occurring in patients who have free aortic regurgitation but no mitral stenosis It is impossible to make the diagnosis in the presence of rheumatic heart disease, and it can be made with confidence only in patients who have syphilitic aortic regurgitation and do not have an accentuated first sound or an opening snap

MITRAL DIASTOLIC MURMUR IN THE PRESENCE OF A RUPTURED PAPILLARY MUSCLE In 1953 Craddock and his associates reviewed 43 cases of ruptured papillary muscles and stated that diastolic murmurs were noted in 5 and systolic murmurs in 19

Loud apical systolic murmurs occurring after acute anterior myocardial infarction in patients in whom these murmurs had not been present previously should suggest this diagnosis

VIRTUAL OR RELATIVE MITRAL STENOSIS In 1897 Rolleston and Dickinson[6] reported 3 cases in which postmortem examination revealed all the anatomic details of mitral stenosis but on such a scale that true stenosis, by measurement did not exist The valve segments were thickened and welded together so as to form a funnel, but the actual orifice was as large as, or larger than, normal and the left ventricle was greatly dilated

The apical systolic murmur of mitral regurgitation was present and frequently a mitral presystolic murmur also

LESIONS CAUSING OBSTRUCTION OF THE MITRAL VALVE ORIFICE

Lesions other than mitral stenosis which cause mitral valve obstruction are rare They include ball valve thrombi or tumors of the left atrium, foreign bodies such as shrapnel in the valve ring and the rare case of external compression due to constrictive pericarditis with severe calcium deposits in the left atrioventricular groove

BALL VALVE THROMBI AND TUMORS IN THE LEFT ATRIUM (FIG 68) Ball valve thrombi or tumors such as myxoma in the left atrium may cause apical diastolic murmurs which cannot be distinguished from those of mitral stenosis except that an opening snap is rarely encountered

There are some auscultatory clues to the diagnosis in those cases where the mass lies free in the left atrial cavity The heart sounds and murmurs may vary in quality and in intensity from beat to beat or at varying interval depending on the degree to which the mass obstructs the orifice of the valve and interferes with its function In addition, clicking sounds may be scattered irregularly throughout systole and diastole

In contrast in patients whose thrombus or tumor mass is fixed in its relation to the mitral orifice or who have a pedunculated tumor extending through the mitral valve down into the left ventricular cavity, there may be

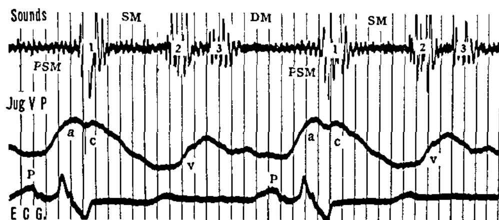


Fig 66 (Apex Log) Mid diastolic and presystolic murmurs in cardiomegaly associated with cirrhosis of the liver secondary to vitamin B deficiency Note the moderate intensity first sound (1) 0 10 sec after the onset of the QRS complex the low frequency low intensity systolic murmur (SM) the normal intensity second sound (2) split by 0 04 sec the mid diastolic vibrations around the timing of the third sound (3) and the presystolic crescendic vibrations (PSM) of low frequency and low to moderate intensity

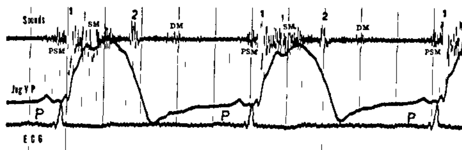


Fig 67 (Apex Log) Mid diastolic and presystolic murmurs in coarctation of the aorta Note the prominent first sound (1) 0 08 sec after the onset of the QRS complex the early crescendo decrescendo systolic murmur (SM) of medium frequency and moderate intensity with the apex of the murmur before mid systole the normal second heart sound (2) the mid diastolic murmur (DM) of low frequency and low intensity and the presystolic murmur (PSM) crescendic up to the first sound

2 Severe systemic hypertension with cardiomegaly but without aortic insufficiency
3 Free aortic regurgitation
4 Occasionally with cardiomegaly following acute myocardial infarction
5 Ventricular aneurysm
6 Adhesive pericarditis
7 Severe anemia (other factors such as increased cardiac output and altered blood viscosity are also important in this group)

ADHESIVE PERICARDITIS Throughout the literature there are numerous accounts of apical diastolic rumbles in patients with adhesive pericarditis In 1894 Fisher reviewed the records of 13 children with this condition Hypertrophy and dilatation of the left ventricle occurred in all 13 at post mortem examination, all had normal mitral valves, yet 5 of the 13 had had apical diastolic rumbles

AORTIC INSUFFICIENCY In 1895 Phear [3] reviewed 46 cases of apical dias

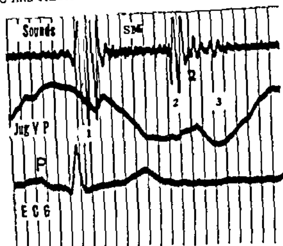


Fig 69 (Apex Log) Vibrations following the second heart sound which could be mis taken for a diastolic rumble Note the loud first heart sound (1) 0 06 sec after the onset of the QRS complex the low intensity medium frequency systolic murmur (SM) the normal second heart sound (2) with the second component much less intense than the first and the low intensity third heart sound (3) The decrescendo second heart sound (2) and the third heart sound (3) together with the vibrations between the second and third heart sounds could be mistaken for an early low frequency diastolic murmur

tion Postmortem examination revealed apparently normal hearts with normal mitral valves

In 1931 Bramwell and Ellis[9] noted either a presystolic murmur or an accentuated first sound at the cardiac apex in 12 Olympic marathon runners They stated that the auscultatory results were identical with those encountered in mitral stenosis

In 1949, Alimurung and his co workers[10] presented 8 cases in which the first heart sound at the apex had been mistaken for a presystolic murmur In all of these cases the first sound had a crescendic configuration on the phonocardiogram There may be many causes for this crescendic effect

1 In some cases the first sound is split with the second part of the split being louder than the first part

2 There may be an early systolic click as seen in some cases of aortic regurgitation or stenosis with a dilated ascending aorta or in pulmonary hypertension If this click is preceded by a broad first sound or by a short systolic murmur then it may not be possible to distinguish these sound from a presystolic crescendo murmur

3 Occasionally the atrial components of the first sound may be more marked than usual and the combination of this followed by a sharp valve closure may simulate a presystolic crescendo murmur

Apical diastolic vibrations in the early stages of rheumatic mitral valvular disease

In 1940 Taquini and his associates[11] described certain phonocardiographic observations in a group of patients with rheumatic heart disease who, after clinical examination, were thought to have diastolic rumbles They



Fig 68 (Apex Log with apex cardiogram) Variations in the rapid inflow wave and in the intensity of the third heart sound in a ball valve thrombus of the left atrium Note the normal first heart sound (1) 0 08 sec after the onset of the QRS complex the decrescendo low intensity systolic murmur (SM) the normal second heart sound (2) the third heart sound (3) louder in the first cycle than in the second and coinciding with a rapid inflow wave (RI) of the apex cardiogram which is more marked in the first cycle than in the second and the systolic clicks (SC) and diastolic clicks (DC) which occur occasionally The diastolic vibrations following the third heart sound are less marked in the second cardiac cycle Muscle tremor accounts for the irregular baseline on the electrocardiogram

no variation in the sounds and murmurs from beat to beat, and the murmur may resemble the classic murmur of mitral stenosis

CALCIFIED PERICARDIUM WITH EXTERNAL MITRAL STENOSIS The association of constrictive pericarditis and rheumatic mitral stenosis is exceedingly rare In the only case in which we made this diagnosis with confidence, operation revealed heavy calcium deposits in the left atrioventricular groove, causing external compression of the mitral ring After successful pericardiectomy the apical mid diastolic rumble, which had been clearly audible prior to operation, disappeared completely

AUSCULTATORY SIGNS IN NORMAL OR SLIGHTLY DILATED HEARTS IMITATING APICAL DIASTOLIC OR PRESYSTOLIC MURMURS (FIG 69)

NORMAL HEARTS WITH A MURMUR CLOSELY RESEMBLING THE PRESYSTOLIC MURMUR OF MITRAL STENOSIS

In 1909, Henry Sewall[7] stated that in structurally normal hearts, especially during excitement, the first heart sound frequently is crescendic, closely simulating either a short apical presystolic murmur, or an accentuated first heart sound similar to that of organic mitral stenosis

It is possible that in some cases of dilatation of the left ventricle, imperfect closure of the mitral valve may result from dilatation of the mitral ring, or the mitral valve leaflets may close completely only after systole has been in progress for a short time This could explain a regurgitant murmur early in systole, before the actual occurrence of the first sound, which may be delayed, or its early components may be masked by the murmur

In 1922, Irons and Jennings[8] reported 4 cases diagnosed as mitral stenosis during life because of a short presystolic murmur heard on ausculta

9 Bramwell C and Elli R Some observations on the circulatory mechanism in mara thon runners Quart J Med 24 329 1931
10 Ahmurung M M Rappaport M B and Sprague H B Variations in first apical sound simulating o-called presystolic murmur of mitral stenosis Phonocardio graphic study New England J Med 241 631 1949
11 Taquini A C Massell B F and Walsh B J Phonocardiographic studies of early rheumatic mitral disease Am Heart J 20 295 1940

stated that a patient with rheumatic mitral valve involvement of less than one year's duration, always has an apical systolic murmur of at least moderate intensity, and frequently, in addition, has a moderately loud extra heart sound (third sound) or a rumbling murmur in diastole Sometimes, prolongation of the vibrations of the third sound produced the acoustic effect of a rumble, whereas, at other times the vibrations of an atrial sound occurring shortly after, or superimposed on, the prolonged vibrations of a third heart sound enhanced the acoustic effect of a rumble

A brief consideration of this paper brings up two questions which cannot be answered easily (1) When does a prolonged third sound become a rumble? (2) When do atrial sound vibrations become a presystolic murmur?

It would seem that since most of these patients did have mitral insufficiency, the blood flow from the left atrium to the left ventricle during diastole would be greater than normal This increased filling early in diastole would undoubtedly give rise to a loud third heart sound in some patients and to a third sound together with a true rumble in others Obviously there must be patients in whom a strict dividing line is impossible The same difficulty would arise with atrial sounds and presystolic murmurs

SUMMARY

This discussion of some of the many causes of apical diastolic murmurs seems to suggest that the presence of such a murmur is by no means sufficient evidence for a diagnosis of mitral stenosis A careful clinical history, the age of the patient, and all accessory information which is available may be necessary for an accurate appraisal There are some cases in which, in spite of all presently employed methods of examination, the condition of the mitral valve cannot be evaluated without direct observation, either at surgery or at postmortem examination

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8 Irons E E, and Jennings A F Presystolic murmurs in rapid hearts simulating murmur of mitral stenosis with report of necropsies J A M A *78* *957* *1922*

section III

diseases of the heart valves

12 / Mitral Valve

MITRAL INSUFFICIENCY

MR

CAUSES OF APICAL SYSTOLIC MURMURS OF MITRAL INSUFFICIENCY

ACUTE RHEUMATIC MYOCARDITIS Generalized cardiac dilatation with involvement of the mitral ring as well as the acute inflammatory process affecting the valves themselves may accompany acute rheumatic heart disease. This subject is discussed more fully on pages 234, 235

CHRONIC RHEUMATIC HEART DISEASE (SEE PP 240 244) Permanent damage to the mitral valve, causing mitral insufficiency, is a frequent sequela of acute rheumatic carditis This damage, though permanent in the sense that the valves never return to normal, may, nevertheless, change in degree as the years go by and mitral insufficiency may become more or less marked, or mitral stenosis may develop and, in time, may become the dominant lesion

MYOCARDITIS FROM VARIOUS OTHER CAUSES Dilatation of the left ventricle, with stretching of the mitral ring as part of the pathologic process, may occur after almost any form of severe myocarditis and insufficiency occurs if the valve leaflets are not adequate to cover the enlarged mitral orifice

DILATATION AND FAILURE OF THE LEFT VENTRICLE Mitral insufficiency may result from dilatation and failure of the left ventricle, regardless of its cause The more common causes are

1 Hypertension either essential or secondary to conditions such as nephritis or coarctation of the aorta
2 Aortic stenosis
3 Myocardial fibrosis associated with disease of the coronary arteries or with long standing anemia
4 Acute cardiac failure following myocardial infarction or sudden loss of blood
5 Luetic or rheumatic aortic regurgitation
6 Thyrotoxicosis

Endocarditis acute or subacute usually secondary to rheumatic or congenital lesions

Bacterial endocarditis causes the most striking deformities of the mitral valve The rapid growth of massive vegetations in acute bacterial endocarditis, as well as the slower development in the subacute form results in regurgitation from interference with valve closure and also from actual

accounted for by the associated left ventricular dilatation or the increased left ventricular inflow in early diastole The systolic murmur may or may not be accompanied by a thrill

RUPTURE OF A PAPILLARY MUSCLE Rupture of a papillary muscle is much more serious than rupture of the chordae tendineae It rarely occurs in the right ventricle, and when it does it is due to endocarditis Rupture of a mitral papillary muscle almost always follows myocardial infarction, and since the cardiac reserve is poor in these patients, acute pulmonary edema or peripheral circulatory collapse results The posterior muscle is twice as commonly affected as the anterior [1]

This lesion may be accompanied by apical systolic or diastolic murmurs, or both Some patients have been described in whom there were no murmurs, and considering the state of collapse and the reduced cardiac output associated with the infarct, this is not surprising

The history of sudden onset, associated with the clinical findings of an acute myocardial infarction and the precarious clinical status of the patient, should help to differentiate this condition from that of rupture of the chordae tendineae However, it has also been reported as due to syphilis, trauma, and polyarteritis nodosa

CALCIFICATION OF MITRAL VALVE LEAFLETS Calcification is often seen in the mitral valve leaflets of patients who have rheumatic heart disease If the calcification is extensive, the free margins of the valves are often involved, whereas if the deposits are smaller, it is usually the body of the leaflets or the adjacent left atrial wall which is affected There is almost always considerable deformity of the mitral valve, and, while the predominant lesion is usually mitral stenosis, there may also be considerable mitral insufficiency A clinical point which may be confusing is that even with tight mitral stenosis, the first heart sound may be normal or diminished in intensity because of the fixed, fibrotic, and calcified mitral valve If an apical systolic murmur is present, the clinician may well be misled into making a diagnosis of predominant mitral insufficiency Even when the valve orifice is rigid, and unable to close mitral insufficiency may not be present because the mobility of the cusps may permit apposition of the leaflets during ventricular systole

CALCIFICATION OF MITRAL RING Occasionally, an apical systolic murmur (usually loud, sometimes harsh, and sometimes rather musical in character) occurs in persons beyond middle age Such persons may have calcification of the mitral ring Simon and Liu observed this calcification at postmortem examination in 10 per cent of 590 persons more than fifty years of age Approximately one fourth of these had associated aortic valvular calcification approximately one fourth had some degree of dilatation or hypertrophy of the left atrium, and in two thirds there was an apical systolic murmur, usually described as loud, rough, or musical

It is believed that extensive calcification of the mitral ring interferes with

ulceration or perforation of the cusps or rupture of chordae Soft systolic murmurs are common in infectious states, but the appearance of a loud murmur in the presence of known bacteremia usually indicates a destructive process at the valve Subacute bacterial endocarditis *is much more frequently* associated with rheumatic mitral insufficiency than with high grade stenosis, therefore, a systolic murmur is usually present for years before the bacterial complication occurs However, acute bacterial endocarditis may involve previously normal valves During the course of the bacterial endocarditis, mitral diastolic murmurs related to increasing obstruction of the valve by the vegetations may appear

FEBRILE STATES Any condition causing a sufficient increase in heart rate and stroke volume may produce systolic murmurs not ordinarily present Frequently these murmurs can be recorded best at the pulmonic area, but they may be heard well at the cardiac apex, and their exact cause is not known When cardiomegaly is present, mitral regurgitation secondary to a dilated mitral ring could be the cause, but this does not represent the majority of cases Whatever the cause, the clinician is advised to be wary of placing too much emphasis on the presence of murmurs under these conditions

APICAL SYSTOLIC MURMURS APPEARING AFTER EXERCISE In the great majority of healthy persons, moderate to vigorous exercise results in the temporary production of clearly audible apical systolic murmurs It is our belief that the appearance of an apical systolic murmur after exercise, where none has been audible previously, is probably of no clinical significance

PREGNANCY During pregnancy, several factors, each of which could produce a murmur independently, are combined in one patient These factors include persistent tachycardia, change in cardiac position, alterations in the volume and composition of the blood, and increased cardiac output

NEUROCIRCULATORY ASTHENIA Apical or basal cardiac systolic murmurs are often encountered in this condition, and attributing these murmurs to organic heart disease can do endless harm

RUPTURE OF THE CHORDAE TENDINEAE OF THE MITRAL VALVE This relatively uncommon event has been reported to occur spontaneously as well as following bacterial endocarditis, severe external trauma to the chest, and prolonged rheumatic fever

The severity of the mitral regurgitation which follows the rupture depends on which leaflet of the mitral valve is affected and on the degree to which it is affected If the lesion involves the anteromedial or aortic leaflet of the mitral valve, the degree of regurgitation is much greater than when the posterolateral or ventricular leaflet is involved

Usually, the murmurs are harsh and blowing, loud in intensity, and maximal at the apex and across to the lower left sternal border As would be expected, they are conducted well to the left axilla and left lung base Occasionally, apical mid diastolic murmurs are also heard, these may well be

criteria for the diagnosis of mitral insufficiency are not adequate If a loud murmur (grade two plus or louder) which is blowing in character, lasts throughout systole, and is well conducted to the left axilla and left lung base, is used as a criterion, many cases of mitral regurgitation will be missed However, since mitral regurgitation is well tolerated in it lesser degrees, less harm will be done by overlooking these cases, than by labeling all apical systolic murmurs as indicative of significant mitral insufficiency A full consideration of such factors as the quality intensity, and transmission of the murmur will enable a more accurate diagnosis and a better appreciation of the prognosis

Heart sounds

First heart sound In the presence of pure mitral insufficiency the first heart sound may be diminished, but with associated mitral stenosis it may be normal or accentuated If mitral insufficiency is secondary to acute rheumatic fever there may be a further diminution in the intensity of the first heart sound especially if first degree heart block is present When mitral insufficiency is present, phonocardiographic analysis of the first sound reveals that in many cases where a diminished first heart sound had been noted clinically the intensity had actually been normal but had been masked by the loud systolic murmur

Second heart sound Since mitral insufficiency is often associated with *elevated left atrial*, pulmonary capillary, and pulmonary artery pressures, it is not unusual to encounter an accentuated second sound at the pulmonic area A well-split second sound may be noted, possibly caused by shortening of left ventricular systole with early closure of the aortic valves

Third heart sound Because of the increased left atrial blood volume and the increased left atrial pressure, early diastolic filling occurs under conditions which are an exaggeration of those factors normally contributing to the production of the physiologic third sound Increased diastolic flow under increased pressure frequently results *in a loud third* heart sound when there is only a moderate degree of mitral insufficiency

Evaluation of mitral regurgitation

The accurate assessment of mitral insufficiency in patients requiring operation for the relief of mitral stenosis is particularly important While it is true that mild or even *moderate degrees* of mitral insufficiency do not contraindicate mitral commissurotomy and although there have been some reports of the mitral insufficiency being lessened after this operation, nevertheless severe degrees of mitral *regurgitation* increase the immediate operative risk and diminish the chances of a favorable long term prognosis

In recent years most studies of mitral regurgitation have been based on the ready acceptance of the *surgeon's* ability to detect this condition at operation if it is present and to rule it out if it is not Hence the percentage

the usual systolic contraction and diastolic relaxation of the ring thought to occur under normal conditions As a result, the mitral leaflet cannot close completely, and mitral regurgitation results The lesion is thought to be atherosclerotic in origin It should be noted that the mitral valve leaflets are relatively normal in this condition, as opposed to rheumatic heart disease, where they may be considerably deformed

Kantrowitz[3] devised a bloodless heart preparation which demonstrated the importance of the annulus of the mitral valve and emphasized the remarkable amount of movement which occurs at the valve ring itself During ventricular systole, there is a rhythmic sphincter like contraction of the entire mitral ring, with considerable reduction in the size of the atrioventricular canal which remains to be closed by the mitral cusps These facts emphasize that both muscular and valvular movements are important in the efficient functioning of the mitral valve, and that calcification of the mitral annulus, preventing its contraction, could result in mitral regurgitation in the presence of normal valve leaflets

MITRAL INSUFFICIENCY IN THE PRESENCE OF MITRAL STENOSIS Sometimes in the presence of severe mitral stenosis, there may be a loud (grade three to four) apical systolic murmur in addition to the typical murmur of mitral stenosis This loud murmur represents mitral insufficiency, but the loudness of the murmur may not indicate the degree of regurgitation Possibly, in such cases, the mitral orifice may be small and fixed in shape, because of the hard, fibrotic, and even calcified valve leaflets Under these conditions, a relatively small regurgitant jet may be accompanied by a loud murmur The situation is somewhat analogous to that of the loud murmur which accompanies the small left to right shunt through a Roger type of ventricular septal defect

PROGNOSIS OF SIGNIFICANT MITRAL SYSTOLIC MURMURS
(SEE APICAL SYSTOLIC MURMURS, P 171)

Apical systolic murmurs indicating mitral insufficiency may be compatible with a long life, with few if any signs of cardiac disability However, more often, the murmur represents some abnormality which if not corrected usually results in a reduction of the patient's exercise tolerance and also may shorten his expected life span

In White's[4] series of 1050 private patients, 270 had loud systolic murmurs, 75 (28 per cent) of these died within a few years of discovery of the murmur Moderately loud systolic murmurs were present in 240 of whom 41 (17 per cent) died within a few years Slight systolic murmurs occurred in 490, and 47 (9 6 per cent) of these died within a few years These patients having been referred to a cardiac consultant, probably had had their murmurs for varying periods prior to being seen by Dr White, but even so, the conclusions are obvious

It is apparent that, as far as apical systolic murmurs are concerned, the

criteria for the diagnosis of mitral insufficiency are not adequate If a loud murmur (grade two plus or louder) which is blowing in character, lasts throughout systole, and is well conducted to the left axilla and left lung base, is used as a criterion many cases of mitral regurgitation will be missed However, since mitral regurgitation is well tolerated in its lesser degrees, less harm will be done by overlooking these cases, than by labeling all apical systolic murmurs as indicative of significant mitral insufficiency A full consideration of such factors as the quality, intensity, and transmission of the murmur will enable a more accurate diagnosis and a better appreciation of the prognosis

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Third heart sound Because of the increased left atrial blood volume and the increased left atrial pressure, early diastolic filling occurs under conditions which are an exaggeration of those factors normally contributing to the production of the physiologic third sound Increased diastolic flow under increased pressure frequently results in a loud third heart sound when there is only a moderate degree of mitral insufficiency

Evaluation of mitral regurgitation

The accurate assessment of mitral insufficiency in patients requiring operation for the relief of mitral stenosis is particularly important While it is true that mild or even moderate degrees of mitral insufficiency do not contraindicate mitral commissurotomy, and, although there have been some reports of the mitral insufficiency being lessened after this operation, nevertheless severe degrees of mitral regurgitation increase the immediate operative risk and diminish the chances of a favorable long term prognosis

In recent years most studies of mitral regurgitation have been based on the ready acceptance of the surgeon's ability to detect this condition at operation if it is present, and to rule it out if it is not Hence the percentage

figures quoted as an index of the accuracy of the different methods of investigation are compared with a standard established by the surgeon's palpating finger Although the latter is our best available confirmatory evidence of mitral insufficiency, it suffers from several obvious limitations Among these are the variations in skill and experience of different surgeons, the sensitivity of their palpating fingers, and the altered physiologic conditions These latter include a generally lowered systemic pressure and, especially, a lowered left atrial pressure Since the degree of mitral insufficiency must be related to the pressure differences between the left ventricle and the left atrium during ventricular systole, as well as to the degree of valvular incompetence, it is obvious that any significant changes in the heart rate or blood pressure may affect the degree of insufficiency

While it is said that left ventricular enlargement associated with mitral valve disease indicates mitral insufficiency when there is no aortic valve disease, hypertension, rheumatic myocarditis, or left ventricular failure, this statement requires some qualifications To rule out the presence of hypertension is simple, but the assessment of the other factors in the presence of severe mitral stenosis can be one of the most difficult problems in differential diagnosis If Aschoff bodies can be taken as an indication of an active rheumatic process, then we know from numerous studies of cardiac muscle obtained at biopsy that low grade active rheumatic fever cannot be excluded by any presently known clinical or laboratory examination The assessment of aortic valve disease in the presence of severe mitral stenosis with diminished cardiac output is exceedingly difficult Patients have been known to have been operated on for tight mitral stenosis, which indeed they had, only to die in the immediate postoperative period from previously unsuspected severe aortic stenosis which was not revealed until postmortem examination

In older patients who have mitral stenosis and aortic stenosis, or myocardial fibrosis with associated coronary artery disease, the estimation of the individual importance of these lesions may be impossible without catheterization of the left side of the heart

FINAL ASSESSMENT OF MITRAL INSUFFICIENCY

Of the various methods discussed in the evaluation of the degree of mitral regurgitation, it would seem that the most useful, from the practical point of view, are the apical systolic murmur, an enlarged left atrium as revealed by x ray, definite systolic pulsation of the left atrium at fluoroscopy, and an electrocardiogram showing left ventricular hypertrophy or combined left and right ventricular hypertrophy, with or without the signs of incomplete right bundle branch block It is obvious that the more of these criteria which are present in addition to the "significant" murmur, the more accurate will be the final diagnosis

MITRAL STENOSIS

THE DIASTOLIC MURMUR OF MITRAL STENOSIS

DESCRIPTIVE HISTORY A review of the descriptive history of the diastolic and presystolic murmurs of mitral stenosis indicates that during the past fifty years little has been added to knowledge of their cause and only minor points have been contributed to the clinical description

In 1841, Gendrin[5] first used the term presystolic Two years later, Fauvel[6] demonstrated that the murmur of mitral stenosis was presystolic, Herard[7] who closely followed Fauvel in his writings, seems to have been well aware of the effects of atrial fibrillation and congestive heart failure on the murmurs of mitral steno is Hope[8] early recognized that the intensity of an apical diastolic murmur did not necessarily indicate the degree of mitral stenosis He stated that " murmurs are not, as is often supposed, louder, *caeteris paribus* in proportion as the valvular contraction is greater On the contrary the loudest murmurs are produced by a moderate contraction, and they become weak when it is extreme a contraction of the mitral or tricuspid valve to only two three, or four lines [one line = 1/12 inch] in diameter, I have frequently known to occasion little or no murmur" Williams[9] distinguished the diastolic murmur of mitral stenosis from that of aortic regurgitation and Markham[10] described the loud apical first heart sound in some cases of mitral stenosis Later, Gairdner,[11 12] of Scotland, introduced the concept that the presystolic part of the murmur of mitral stenosis was due to atrial systole

Concurrently with the investigation of Gairdner[11 12] in Scotland Austin Flint[14 1] in the United States, was following a similar line of thought and was writing extensively on the presystolic murmur, but he paid little attention to the murmur occurring during mid diastole

VARIOUS FACTORS AFFECTING THE MURMURS *Effect of change of rhythm on the presystolic murmur* Fagge was one of the first to note that the presystolic murmur could be altered by a change in rhythm He reported a case in which the heart was liable to sudden changes in rhythm, from regularity to a bigeminy with probable dropping of every third beat and corresponding changes in the mid diastolic and presystolic murmurs

Effect of first degree heart block on the presystolic murmur Galabin[23] using a modification of Marey s cardiograph found in 1875, that in certain cases the diastolic murmur began shortly after the second sound and was separated by a short pause from the first sound (Figs 70 to 72)

Effect of second and third degree heart block on the diastolic murmur Many years after Galabin[23] Sir Thomas Lewis[4] pointed out the effect of atrial systole on the mitral diastolic murmur in cases of heart block, noting that a murmur occurred with each atrial contraction (Fig 73)

Effect of atrial fibrillation on the presystolic murmur There is no doubt

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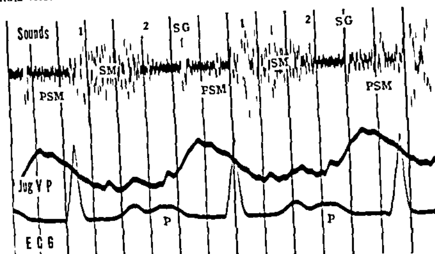


Fig 72 (Ap x Log) Phonocard ogram of 19 year old g rl with active rheumat c fe er (th rd attack) m tral regurg tat o m t al stenos s aort c regurg tation a d f st degree heart block It ll strates another effect of a l ng P R terval on the card ac sounds The first heart sound is moderately int nse desp te the long P R interval of 0 29 sec The second sound s of rather low intens ty at the apex The th d hea t sound is unusually prom nent and s co s derably louder at the apex than the second hea t sound wh ch is to some extent obscu ed by the systol c murmur (SM) The atrial sou d may be summated w th the th rd he t sou d and is also co c ned w th the murmu (PSM) that fills the terval between the th d and first so nds but w thout presystol c c esce do The systol c m mu s cresce do dec esc ndo a d fills systole The summat o gallop (SG) results from an accentuat on of the no m l th d hea t so nd as a r s lt of relat ely ea ly atrial contract on secondary to the prolo ged P R nterval (From O gley P A Sp ag e H B and Rappaport M B New England J Med 253 1049 1955)

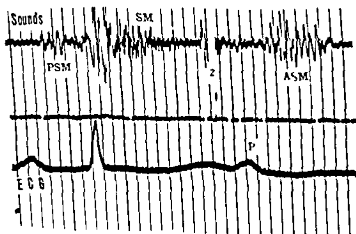


Fig 73 (Ape Log) Ap cal t c gs n 65 y ar old woma w th a lo g h story of hyp t s o and c mplete hea t block Th e w s no defin te h story of rheumatic feve The pho oca d og am d mo str t s th fact that atr al systole is capable of caus ng a loud t al ystol c m m r (PSM) and (ASM) a d th t its pos t on in d astole va ies w th the pos t o f th P wa e of th elect oca d og am (F om O gley P A Sp ague H B a d R ppapo t M B N w England J Med 253 1049 1955)

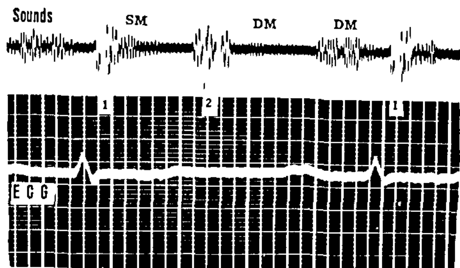


Fig 70 (Apex Log) Phonocardiogram of 44 year old woman with a history of rheumatic heart disease since childhood and considerable limitation of activity during the last few years It illustrates the effect of a long P R interval on the atrial systolic murmur (SM) which be comes late diastolic rather than presystolic The first diastolic murmur (DM) is a rumbling low pitched low intensity murmur occurring around the time of the normal third heart sound The second diastolic murmur (DM) is equivalent to an atrial systolic murmur that follows the P wave of the electrocardiogram (E C G) and is separated by a considerable interval from the first heart sound (From Ongley P A Sprague H B and Rappaport M B New England J Med 253 1049 1955)

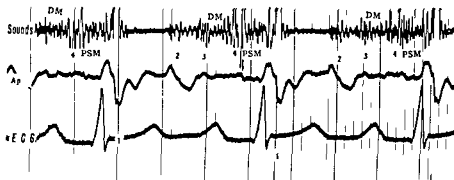


Fig 71 (Apex Log) Phonocardiogram of 22 year old man with rheumatic heart disease and mitral stenosis illustrating the fact that with intense atrial activity and a slightly pro longed P R interval (0 26 sec) a presystolic gallop (4) may be present together with a presystolic murmur (PSM) which is thus not crescendo in type If the P R interval had been normal *the murmur would doubtless have been a presystolic crescendo The earlier part of the* diastolic murmur (DM) becomes evident at the time of the third heart sound (rapid ven tricular filling) although it starts before it at the opening of the mitral valve (From Ongley P A Sprague H B and Rappaport M B New England J Med 253 1049 1955)

that a murmur can be heard in presystole in cases of mitral stenosis with atrial fibrillation when diastole is short This murmur is not the atrial sys tolic murmur described by Gairdner [11 13] but is due simply to the falling of the first heart sound earlier than usual in diastole because of the rapid heart rate If the rate slows, or if there is a long diastolic pause, the diastolic murmur will be found to fade off in a decrescendo fashion (Figs 74 and 75)

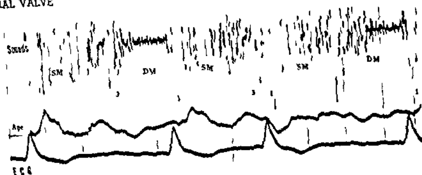


Fig 75 (Apex Log) Apical tracings in 32 year old woman with rheumatic heart disease mitral stenosis mitral regurgitation aortic regurgitation (slight) and atrial fibrillation The phonocardiogram demonstrates the great variability in the diastolic murmur in relation to cycle length In the first and third cycles the murmur runs throughout diastole and is of moderate intensity In the second cycle the third sound is followed so rapidly by the first sound of the next cycle that a crescendo effect results on auscultation (From Ongley P A Sprague H B and Rappaport M B New England J Med 253 1049 1955)



Fig 76 (Apex Log) Apical tracing illustrating the absence of the presystolic murmur in the ventricular ectopic beat and also after the compensatory pause (From Ongley P A Sprague H B and Rappaport M B New England J Med 253 1049 1955)

Effect of the Valsalva maneuver on the diastolic murmur of mitral stenosis It has been shown that the mitral diastolic murmur can be affected in many different ways depending on variations in cardiac rhythm It is also possible to alter the murmur considerably by such a simple maneuver as forced expiration against a closed glottis (Valsalva's maneuver) The positive pressure that develops within the thorax decreases venous return and in turn decreases flow through the mitral valve orifice The enlarged left atrium continues to beat forcibly, and a presystolic murmur may be converted into an atrial sound giving a presystolic gallop (Fig 77)

Effect of exercise and change of position on the diastolic murmur of mitral stenosis Friedrich [6] stated in 1867 that a dull or reduplicated diastolic tone frequently could be converted into a distinct diastolic murmur when a patient was made to walk up and down to accelerate the heart's action

Bramwell [7] in 1881 further observed that "in some cases of mitral stenosis the murmur disappears when the position of the patient is altered, when he gets up but the rhythm of the murmur does not change, it always remains presystolic The subject of the effect of change in position (Fig 78) was more fully studied by Gowers [8]

The best description of this phenomenon occurs in Sir James MacKenzie's detailed discussion of the subject in 1913 [2]

Effect of ventricular premature beats on the presystolic murmur It is obvious that no atrial systolic murmur will be heard before the first heart sound of a ventricular premature beat, since there is no preceding atrial contraction It is interesting that the presystolic murmur resulting from atrial contraction may also be absent in the beat that follows the ventricular premature beat A possible explanation for this is the fact that, owing to the long compensatory pause, the left ventricle may become filled with blood so that the atrium cannot push enough extra blood through the mitral orifice in late diastole to create a murmur (Fig 76)

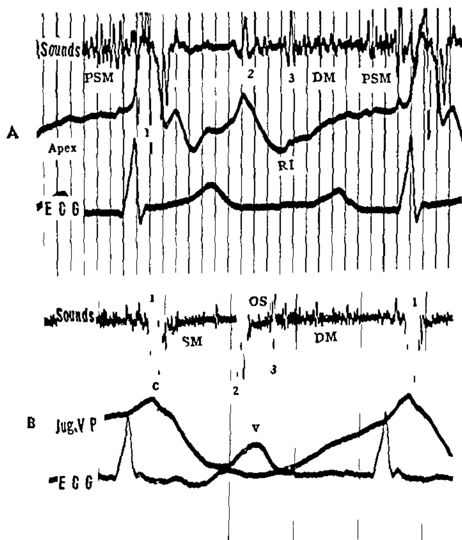


Fig 74 A (Apex Log) Phonocardiogram of 32 year old man demonstrating a presystolic crescendo murmur (PSM) when normal rhythm is present RI indicates rapid inflow B (Apex Log and jugular venous pulse) Phonocardiogram of same patient after the onset of atrial fibrillation Note that the diastolic murmur still extends into presystole but there is no crescendo effect (From Ongley P A Sprague H B and Rappaport M B New England J Med 253 1049 1955)

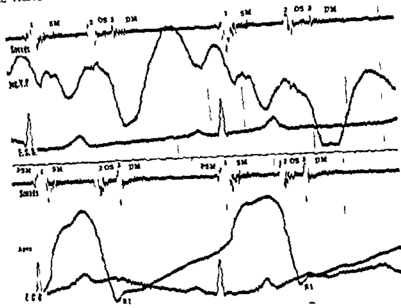


Fig 78 Apical tracings showing the effect of exercise on the diastolic events in a case of mild mitral stenosis Upper tracing (Apex Log and jugular venous pulse) The second sound can be seen followed by an opening snap and then by a third heart sound and a short diastolic murmur which owing to the slow heart rate is early diastolic rather than mid diastolic A slight presystolic murmur is also present Lower tracing (Apex Log) After exercise the only significant alteration is in the greatly increased intensity of the third heart sound For some reason the opening snap has lessened in intensity and would doubtless be inaudible The presystolic murmur has altered very little (From Ongley P A Sprague H B and Rappaport M B New England J Med 253 1049 1955)

was not until 1901 that Broadbent[?] called attention to the disappearance of the presystolic murmur in failure and presented possible causes for this, the most probable one being the establishment of tricuspid incompetence. The giving way of the tricuspid valve and the occurrence of considerable reflux into the right atrium make it impossible for the right ventricle to sustain the high pressure in the pulmonary circulation and the left atrium that was present previously There is not therefore, sufficient pressure to force the blood through the mitral orifice rapidly enough to generate a murmur

Despite the many brilliant clinical observations concerning the presystolic murmur, the theory of its causation and indeed of its actual existence was by no means universally accepted

Ormerod[3] in 1864 disputed the atrial systolic origin of the murmur described by Gairdner[11,13] on the grounds that the atrial contraction was too weak and brief to cause so loud a sound, which he held to be due rather to the contraction of the ventricle and to be regurgitant

Barclay[21] eight years later maintained that the so-called presystolic murmur was really systolic and that although indicative of a contracted

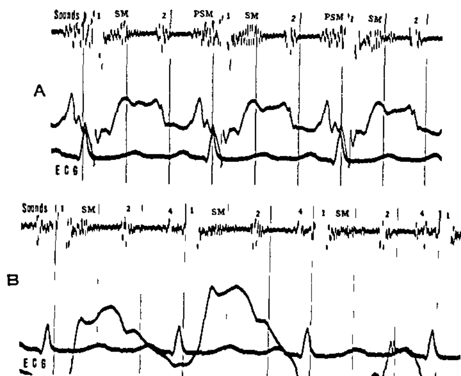


Fig. 77. (Apex Log.) Apical tracing showing presystolic murmur (PSM) before the Valsalva maneuver (A) and (B) alteration of the presystolic murmur to an atrial sound (4) giving a presystolic gallop secondary to the Valsalva maneuver. (From Ongley, P. A., Sprague, H. B., and Rappaport, M. B.: New England J. Med. 253:1049, 1955.)

Effect of mitral valve surgery on the diastolic murmur of mitral stenosis. Mitral valve surgery may have no effect whatsoever, or it may affect the diastolic murmur of mitral stenosis in many ways. If there is a very slight murmur before operation, because of tight mitral stenosis, this murmur may greatly increase after operation because of the increased blood flow over the roughened and torn valve margins. Moreover, a murmur of moderate to loud intensity may be decreased after operation if the split is relatively smooth and no increased vibration results. In some cases the murmur is relatively quiet for a few weeks to a few months after operation and then seems to increase in intensity, perhaps owing to the development of further stenosis. In some patients who have had fibrillation before operation, a return to normal sinus rhythm after operation may be accompanied by the return of a presystolic crescendo murmur. Conversely, in patients who have normal sinus rhythm and who have fibrillation in the first few days or weeks after operation, the atrial systolic murmur will disappear. The development of other bizarre rhythms will, of course, alter the murmur in various ways. Thus, it is seen that the effects of mitral valve surgery on the diastolic murmur may vary greatly, as one would expect (Fig. 79).

Disappearance of the presystolic murmur during congestive heart failure. Although in 1854 Hérard[7] had described the fact that the diastolic murmur of mitral stenosis sometimes disappears during congestive heart failure, it

due to the combined effect of mitral regurgitation occurring very early in systole and followed by a delayed first sound

OPENING SNAP OF THE MITRAL VALVE The opening snap of the mitral valve may be heard in many cases of mitral stenosis It is due simply to an accentuation of the normal fourth component of the second heart sound—that is, the opening of the atrioventricular valves. It was first described by Duroziez[3, 33] in 1862, when he introduced his onomatopoeia, "ffouttatarou" The "ffout" corresponds to the crescendo presystolic murmur and ends abruptly in a snapping first heart sound, the "tata" refers to the second heart sound closely followed by the opening snap of the mitral valve, and the "rou" to the low pitched diastolic rumble The actual term "opening snap" or its French equivalent, was introduced by Rouches[34] in 1888 as "claquement d ouverture de la mitrale"

A third sound coming close after the second sound may be difficult to differentiate clinically from the opening snap, but usually the opening snap, being of higher frequency than the third sound, has a high pitched, dry quality whereas the third sound is dull and low pitched.

Another difficulty occasionally encountered is the differentiation of an opening snap from a split second sound There is a very short interval between the two components of a split second sound, and the components are usually of similar intensity to the ear No reliance can be put on the fact that a split second sound is said to be heard best at the base and an opening snap of the apex Margolies and Wolferth,[3] in 1932, pointed out that the opening snap is often best heard at the base, and we have also recorded this fact on a number of occasions

The phonocardiogram often helps differentiate a split second sound, a second sound combined with an opening snap, and a second sound together with a third heart sound or any combination of these three sounds If a good tracing of an apex cardiogram can be obtained simultaneously with the sound tracing differentiation is easy However a good apex cardiogram is not always obtainable, and time intervals may then be of some assistance

In relation to the apex cardiogram the split second sound has its second component before the 0 point (opening of the atrioventricular valves) An opening snap is synchronous with the 0 point, and a third sound occurs at the summit of the rapid inflow wave (Fig 80)

So far as time intervals are concerned, the duration between the two components of a split second heart sound is usually less than 0 07 sec. The interval between the beginning of the second sound and the opening snap is at least 0 08 sec., and that between the beginning of the second sound complex and the third sound is about 0 12 sec

It is important to realize that the opening snap is one of the few signs in mitral stenosis that may persist even when the murmurs become equivocal or absent, as during atrial fibrillation or failure, or both

In 1951 Messer and his associates[36] studied the interval between the

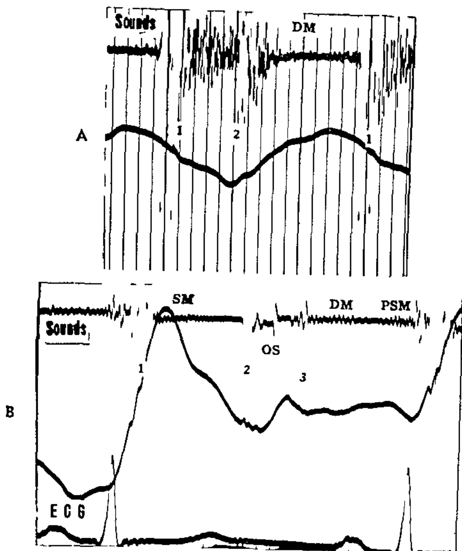


Fig 79 (Apex Log) Apical tracings in a 37 year old woman with rheumatic heart disease mitral stenosis and atrial fibrillation A Before operation in 1951 the patient had a very loud systolic murmur just medial to the apex and the apical diastolic murmur could not be identified with any degree of certainty It was thought that the systolic murmur might have been due to tricuspid incompetence B Two years after operation the systolic murmur of tricuspid regurgitation has disappeared The rhythm has reverted to normal an opening snap (OS) third heart sound (3) diastolic murmur (DM) and presystolic with slight crescendo (PSM) are now easily identified This patient was converted from a cardiac cripple to a relatively normal active healthy woman (From Ongley P A Sprague H B and Rappaport M B New England J Med 253 1049 1955)

mitral orifice, it was regurgitant He called attention to the absence of valves in connection with the pulmonary veins, pointed out that with no means of closing the outlet backwards, the atrium could expand but had little power to drive the blood forward, and claimed that it "was scarcely possible that one of the loudest and roughest murmurs ever heard in cardiac disease should be produced by contraction of the auricle"

The suggestion has been made that the presystolic murmur heard in cases of mitral stenosis with atrial fibrillation is really a systolic murmur and is

Usefulness of the opening snap in estimating the severity of mitral stenosis The atrioventricular valves open when the pressure in the left ventricle falls below that of the left atrium Depending on the left atrial pressure and the rigidity of the mitral leaflets, the opening snap occurs at variable intervals soon after the closing of the semilunar valves If there is severe mitral stenosis with elevated left atrial pressure, the period of isometric relaxation is shortened, and the 2 O S interval is correspondingly diminished With lesser degrees of stenosis, left ventricular pressure must fall to lower levels before the mitral valve opens, and the 2 O S interval is correspondingly prolonged

Although the 2 O S interval indicates the pressure gradient across the mitral valve at the beginning of diastole, it gives no indication of the mean diastolic gradient and there is no way of calculating just how much of the delay in opening may be due to mechanical inertia of the thickened and stiffened valves Bayer and his associates[37] studied 100 cases of pure or predominant mitral stenosis in an endeavor to correlate the pulmonary capillary pressure obtained at cardiac catheterization with the interval between the beginning of the second heart sound and the first vibrations of the opening snap of the mitral valve Of course, in carrying out this type of study it is essential that the pulmonary capillary pressure, which is used to reflect left atrial pressure, and the phonocardiogram be recorded simultaneously, since left atrial pressure and the timing of the opening snap vary with different heart rates and varying cardiac outputs

It was assumed that the interval between the closure of the aortic valve and the opening of the mitral valve would be dependent on the rate of pressure decrease in the left ventricle and the height of the pressure in the left atrium Since the fall of left ventricular pressure is more gradual toward the end of isometric relaxation it is apparent that elevations of left atrial pressure will be more significant in this range Bayer and his co-workers[37] believed that there was a definite relationship between the time interval from the onset of the second sound to the opening snap, and the height of the mean pressure in the left atrium, as shown by the pulmonary capillary pressure They also believed that an increase in left atrial pressure accompanying exercise could be inferred from a shortening of the interval between the onset of the second sound and the opening snap

Since aortic blood pressure is the dominant factor in determining the time of aortic valve closure these studies must be considered in relation to the aortic pressure It is obvious that with higher aortic pressure the aortic valve closes sooner than it does if the pressure is low Bayer and his associates believed that there was little change in timing between aortic pressures of 110 to 130 mm of mercury They stated that in the absence of tachycardia a narrow interval (no longer than 0 05 sec) is consistent with a severe degree of mitral stenosis and reflects a valve size of less than 1 cm^2 An interval longer than 0 05 sec does not necessarily mean slight stenosis, and

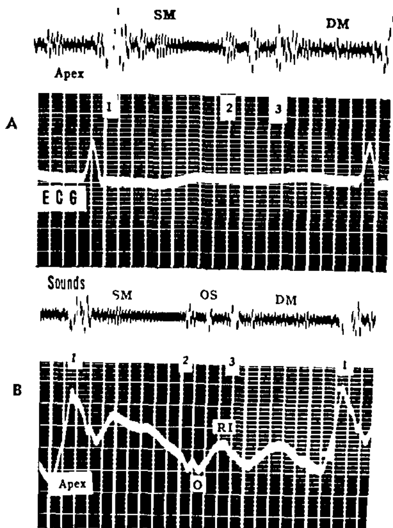


Fig 80 (Apex Log) Apical tracings in a 69 year old woman with atrial fibrillation minimal aortic regurgitation mitral regurgitation and mitral stenosis The phonocardiogram *demonstrates the need for taking an apex cardiogram to be sure whether three sounds* following close together in diastole are due to a split second sound combined with an opening snap or a second sound combined with an opening snap and a third sound In tracing *A with simultaneous electrocardiogram it is not possible to be certain of the origin of the* sound between the second (2) and the third (3) heart sounds However in tracing B the apex cardiogram makes identification easy showing the middle one of the three sounds to *coincide with the valley or O point of the apex cardiogram This is the opening of the* atrioventricular valves and the beginning of the rapid inflow valve wave (RI) hence the inter mediate sound is an opening snap The third sound (3) falls at the apex of the rapid inflow *wave and so is a true third heart sound (From Ongley P A Sprague H B and Rappa* port M B New England J Med 253 1049 1955)

onset of the valvular component of the second heart sound and the opening snap in cases of atrial fibrillation They found that this interval depended largely on the duration of the preceding RR interval They also noted that the opening snap sometimes appeared closer than 0 07 sec to the valvular elements of the second heart sound, emphasizing the importance of relating the duplicated second sound to the apex cardiogram for accurate interpre tation of the nature of this duplication

abnormality of frequency or intensity of the first heart sound However, in many cases of mitral stenosis, no delay can be detected Wells[40] stated that the main components of the first heart sound should occur within 0 06 sec of the onset of the QRS complex and that, if they occur at 0 07 sec or later, the first heart sound should be classified as "late"

Delayed first heart sound in mitral stenosis Delay in the onset of the first heart sound in mitral stenosis has been recognized since the report of Weiss and Joachim[41] in 1911 This delay is thought to be due to the increased left atrial pressure in mitral stenosis, which increases the time required for the isometric contraction of the left ventricle to develop sufficient pressure to exceed left atrial pressure and thereby to close the mitral valve

Attempts have been made by different groups to correlate the delay in mitral valve closure with the severity of the mitral stenosis Certain factors tend to make this correlation difficult

1 The time of onset of mechanical ventricular systole may not have the same relationship to the onset of electrical systole in each patient
2 The rate of increase in pressure in the left ventricle may vary from patient to patient depending on whether or not there is active carditis or long standing myocardial disease
3 If tachycardia is present left ventricular pressure rises at a slower rate than normal
4 The stiffened mitral cusps are more resistant to closure

Kelly[42] studied 75 patients with mitral stenosis and 100 patients with other forms of heart disease in order to correlate the time interval between the onset of the QRS complex in lead II of the electrocardiogram, and the onset of the first rapid vibrations of the first heart sound, with the severity of mitral stenosis Because of the delay in the first sound sometimes associated with bundle branch block, all patients with a QRS interval greater than 0 11 sec were excluded from the study His results are shown in table 15

None of the patients in the first group had a Q 1 interval greater than 0 07 sec whereas in 45 per cent of those who had mitral stenosis the Q 1 interval was greater than 0 07 sec Postoperative studies on some of the

TABLE 15

COMPARISON OF Q-1 INTERVALS IN 100 PATIENTS WHO HAD HEART DISEASE OTHER THAN MITRAL STENOSIS WITH THOSE OF 75 WHO HAD MITRAL STENOSIS AND 25 WHO HAD MITRAL INSUFFICIENCY

Patients Studied	*Average Q 1 Interval* sec	*Standard Deviation* sec
100 patients with heart disease but no mitral stenosis	0 04	±0 01
75 patients with mitral stenosis	0 06	±0 03
25 patients with mitral insufficiency	0 04	±0 01

the effect of exercise on the interval must be considered The degree of shortening of the interval with exercise and the time it takes the interval to return to its pre exercise figure are factors which must be considered in judging the individual patient

Successful mitral valvotomy is said to be followed by an increase in the interval between the onset of the second sound and the opening snap, and the effects of exercise on this interval can be measured easily by the phonocardiograph

Q-1 minus the 2 O S interval Because of the various factors affecting either the Q 1 or the 2 O S interval, Wells [38] believed that consideration should be given to both the delay in the first sound and the 2 O S interval Accordingly, he studied 100 patients between the ages of 18 and 56 years who had predominant mitral stenosis He thought there was a definite relationship between the Q 1 minus the 2 O S interval and the pressure gradient across the mitral valve If the "corrected Q 1 minus 2 O S interval" lies between plus 5½ and minus 1½, the pressure gradient is high, whereas if it lies between minus 1½ and minus 4½, the gradient is low

Variations in the delay of the first sound and the 2 O S interval are found with the changing cycle lengths encountered in patients who have atrial fibrillation associated with mitral stenosis These changes coincide with the length of the preceding cycle In general, after a short cycle, delay in the first sound is accentuated, whereas the 2 O S interval is shortened Conversely, after a long cycle there is less delay in the first sound, and the 2 O S interval is prolonged

With extremely slow rates, any change in the timing of the first sound or in the opening snap must be due to stiffness of the valve itself, since, irrespective of valve size, the left atrium tends to empty and the left ventricle to fill if given sufficient time

FIRST HEART SOUND IN MITRAL STENOSIS Accentuation of the first heart sound, giving it a loud, snapping quality at the apex, is another important and fairly common sign in mitral valve disease It is sometimes the first suggestion of fibrosis and early stenosis but may, of course, result from other conditions, such as exercise, thyrotoxicosis, neurocirculatory asthenia and nodal rhythm There are many cases of long standing mitral stenosis, however, in which the first heart sound is of low intensity or is masked by a systolic murmur that follows immediately Accentuation of the first sound is more indicative of mitral stenosis when found in association with other signs, such as an enlarged left atrium or an accentuated pulmonic second sound

Even when the first sound is not increased in intensity it may be abnormal by phonocardiogram This is shown by a delay of the maximum vibrations of the first heart sound in relation to the QRS complex of the electrocardiogram This was described by Cossio and Berconsky [39] in 1943 and again by Wells in 1952 [40] Wells considered the delay to be more significant than

persons who had had normal Q-1 intervals Normal persons, and presumably those with mitral stenosis, may have different electrical mechanical intervals in the left ventricle (that is the interval between the onset of the QRS in the electrocardiogram and the onset of the increase in pressure in the left ventricle) In persons with no conduction defect this interval averages 0 04 sec, with a range of from 0 02 to 0 06 sec This is the range of the Q 1 interval in patients who do not have mitral stenosis

It is obvious that if a patient with severe mitral stenosis has an electrical mechanical interval of 0 02 to 0 03 sec, a further delay of 0 03 sec for the development of adequate pressure to close the mitral valve would give a Q 1 interval of 0 05 to 0 06 sec, which would fall within the normal range Therefore, it is obvious that a normal Q 1 interval does not exclude severe mitral stenosis

Another important variable to be considered is the rate of increase in left ventricular pressure A study of 12 normal persons showed an increase from 4 to 14 mm of mercury in 0 01 sec[4] One would expect the rate of increase to be slower in patients who have acute myocarditis or long standing myocardial fibrosis

It is not suggested that any of these various measurements be taken as proof of significant mitral stenosis but when correlated with the patient's history, clinical examination roentgenograms and electrocardiogram, they may give valuable leads when the diagnosis is doubtful

Kelly's statement that patients who have congenital heart disease with mid diastolic murmurs and who have a Q 1 interval greater than 0 07 sec have mitral stenosis cannot pass unchallenged It is our experience that the so-called delay in the first heart sound is fairly common in congenital heart disease, whereas mitral stenosis either as an isolated lesion or in combination with other lesions is not common

THIRD HEART SOUND IN MITRAL STENOSIS It has been stressed that all possible aid is required to estimate the degree of disability caused by mitral stenosis In this regard the presence or absence of a third heart sound may be of some assistance Since it is generally accepted that the third heart sound results from the rapid inflow of blood from the left atrium to the left ventricle early in diastole, it is not unreasonable to suppose that as blood flow becomes diminished secondary to tight mitral stenosis the third heart sound progressively lessens in intensity and finally disappears In our limited number of cases we have found this to be true We have also observed the converse—namely, that some patients with typical murmurs of mitral stenosis and good tonus of the left ventricle but with a prominent third heart sound have not been unduly incapacitated by their mitral stenosis supporting the concept that a loud, apical, third heart sound means good filling of the left ventricle As pointed out by Hope[8] over a hundred years ago the moderate cases of mitral stenosis often had the loudest murmur and this may be misleading clinically since a loud mitral diastolic

patients showed no change in the left ventricular diastolic pressure and no change in the electrical mechanical interval of the left ventricle, and yet there was definite shortening of the Q 1 interval, suggesting that the lower left atrial pressure following successful surgery allowed more rapid closure of the mitral valve Comparisons of left atrial and left ventricular pressure curves show that the Q 1 interval corresponds to the expected time of closure of the mitral valve

Because in some cases of suspected mitral stenosis the only murmur heard may be an apical systolic murmur, mitral insufficiency may be con sidered the dominant lesion Since the Q 1 interval is normal in mitral in sufficiency, an apical systolic murmur with a prolonged Q 1 interval should suggest dominant mitral stenosis Whereas it has been shown by Messer and his associates [36] that the Q 1 interval varies with the length of the previous cycle, in patients with atrial fibrillation and mitral stenosis, no such variation was found by Kelly in patients with atrial fibrillation but without mitral stenosis

It is believed that the delay in the Q 1 interval parallels the severity of the mitral stenosis and that patients with a Q 1 interval greater than 0 07 sec have significant mitral stenosis

Of Kelly's 100 patients with mitral stenosis 16 were operated on and studied postoperatively In 15 of these 16 there was shortening of the Q 1 interval

Another useful point in this differential diagnosis may be the presence of a loud third sound in a patient with apical systolic and diastolic murmurs who has a normal Q 1 interval Under these circumstances a loud third sound definitely suggests predominant insufficiency, with increased left ventricular filling early in diastole

Other causes of elevation of left atrial pressure, such as left ventricular failure or constrictive pericarditis do not result in a delay of mitral valve closure, as under these circumstances the left ventricular diastolic pressure also is elevated, and no undue delay is required for left ventricular pressure to exceed left atrial pressure after the onset of isometric contraction

Combined studies of the Q 1 interval and the 2 O S interval (the in terval between the onset of the second sound and the opening snap) cer tainly suggest that any patient who has a long Q 1 interval and a short 2 O S interval has significant mitral stenosis

Wells,[43] using the surgeon's finger as an instrument for estimating the size of the valve, concluded that if the Q 1 interval minus the 2 O S interval had a value greater than minus 0 01 sec , the patient had severe mitral stenosis If the values gave a more negative result than minus 0 015 sec , an orifice greater than 1 sq cm could be expected In general, Kelly [4] confirmed Well's observations, but with some exceptions Only 45 per cent of his patients had Q 1 intervals beyond the control range of 0 02 to 0 06 sec

Postmortem examination sometimes reveals severe mitral stenosis in

DIASTOLIC MURMUR It is essential that the physician carry out his auscultation under ideally quiet conditions

The patient should hold his breath comfortably in moderate expiration As Levine and Harvey [11] have emphasized, the physician must acquire the habit of listening specifically to the individual events of the cardiac cycle He must concentrate on the first sound to the exclusion of all else, then on the second sound, then on systole and finally on diastole There is no other way of accurately timing murmurs and heart sounds or even of being certain of the existence of the lower pitched murmurs and sounds

In listening for the low frequency mid diastolic rumble of mitral stenosis, the bell of the stethoscope is generally more useful than the diaphragm, which tends to screen out the lower frequency sounds so that it may enable those of higher frequency to be heard more easily

When the bell is used, one should apply it first of all firmly to the chest wall and, while listening intently, gradually relax the pressure of the bell against the skin A murmur at first inaudible with firm pressure may become clearly audible with light pressure The reason for this is that firm pressure of the bell against the chest causes the skin to be stretched tightly across the mouth of the bell so that a high frequency diaphragm is created Relaxing this pressure converts the bell to a low frequency recorder, and so the murmur may be heard

Exercising the patient and turning him on his left side, a maneuver known to all physicians but not always employed, should be done in every case

Careful searching in the area of the apex is essential, since, although some murmurs may be heard over a reasonably large area, others are often restricted to small areas of 2 5 to 5 cm in diameter Hence, careful and diligent listening is required in any suspected case if errors are to be avoided

In some cases, the murmur may vary, depending on such factors as heart rate, the presence or absence of atrial fibrillation and congestive heart failure, and the natural progression of the disease Thus, the murmur may be heard by an observer one day and not by the same observer on the next day or even six months or a year or more later Conversely, a patient entering the hospital in failure may have no detectable murmur, and yet, when the rate and rhythm are controlled by digitalis and by bed rest and the failure benefited by drugs, a salt free diet, and diuretics, a mid diastolic murmur, possibly with presystolic crescendo, may become clearly audible These changes, which may occur with varying degrees of valve damage, depend on the relation between the shape of the valve orifice and the rate of blood flow through the orifice and on whether or not turbulence results in the blood stream

If a patient is admitted to the hospital with failure or a condition resembling cor pulmonale and no mitral diastolic murmur is heard, and if the record shows that some previous observer heard a murmur of mitral stenosis, one should not be too hasty in deciding that the earlier observer was in error It is not impossible that as the stenosis has increased the murmur has

murmur frequently suggests to the physician or surgeon that the degree of stenosis is marked It is suggested that the presence of a loud third heart sound in the left ventricle in the presence of the diastolic murmur of mitral stenosis indicates that the degree of stenosis is not unduly severe

FACTORS AFFECTING THE DETECTION OF THE DIASTOLIC MURMUR If a loud systolic murmur is present at the apex or if a loud opening snap or loud second heart sound is heard in the same region, the diastolic murmur may be missed because of fatigue and masking effects on the human hearing mechanism This is especially so in cases in which the diastolic rumble is low pitched or of short duration

The murmur may be missed in the presence of tachycardia, either with regular rhythm or in the presence of atrial fibrillation or flutter

Even though mitral stenosis may be considered to be present clinically this does not mean that the hemodynamics are identical in all cases The valve mechanism, the size of the mitral orifice, the direction of blood flow from atrium to ventricle, the location of the valve orifice (whether centrally or eccentrically placed) and the question whether the valves are moderately or greatly thickened or calcified may all be involved in varying the nature and intensity of the diastolic murmur of mitral stenosis

Especially in patients in the older age group, the presence or absence of heart failure may cause one to miss detecting the murmur This is particularly true in patients with a very tight stenosis

A word of warning is necessary in the assessment of mitral stenosis based on the intensity of the apical diastolic murmur with or without presystolic crescendo Hope [8] pointed out that moderate stenosis gives the loudest murmur and that tight mitral stenosis may exist with little or no murmur Herard [7] observed that in patients in failure or with rapid irregular rhythm, the murmur may be absent Other authors have emphasized the fact that many conditions other than mitral stenosis may be accompanied by an apical diastolic murmur

We have repeatedly observed patients in the older age group with a long history of rheumatic heart disease and the clear cut murmurs of mitral stenosis who suffer very little disability and who can do a full day's work These patients, who often survive to a ripe old age and live full and useful lives (Fig 80), should not be subjected to surgery, which carries a definite risk at any age and a greater risk in the later decades On the other hand, patients in failure, even without a murmur, who have an enlarged left atrium, right axis deviation by electrocardiogram and a history of hemoptysis and perhaps even of rheumatic fever, should be very fully investigated to rule out the possibility of mitral stenosis A loud apical first sound or an opening snap, possibly in the presence of an accentuated pulmonic sound, may be all the auscultatory evidence available to make the diagnosis in these cases

FACTORS IN CLINICAL EXAMINATION WHICH HELP IN DETECTING THE

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lessened until finally it has become inaudible We have frequently encountered this situation In such cases, if the first sound seems too "good," —that is, louder than appears consistent with the degree of myocardial failure—mitral stenosis should be suspected This is especially true if atrial fibrillation and hypertrophy of the right ventricle are present

The presystolic crescendo of the mitral diastolic rumble is not present in all cases of mitral stenosis This fact was pointed out by Sansom,[45, 50] Johnston,[51] and Battro and Braun Menendez[52] Sometimes, the mid diastolic murmur is recorded as diminuendo and may, in fact, almost disappear just before the first heart sound This occurs especially during the long diastole in patients with a slow heart rate or when the atrial contraction is feeble or absent

On the other hand, cases are often reported in which a presystolic crescendo murmur has been heard on auscultation but no abnormality of the mitral valve is detected at postmortem examination Alimurung, Rappaport and Sprague[53] reviewed the causes for this phenomenon in 1949

In any case in which an opening snap is heard, it is well to suspect mitral stenosis, even if no diastolic murmur is heard, especially if the condition clinically resembles mitral stenosis

When a loud snapping first sound is heard at the apex with no obvious cause, such as thyrotoxicosis or neurocirculatory asthenia or nodal rhythm, one should search carefully for a mid diastolic or presystolic murmur However, a quiet or normal first sound at the apex does not exclude the possibility of mitral stenosis

CONCLUSIONS The diagnosis of mitral stenosis may be easy or extremely difficult, and all possible information is desirable in the evaluation of each case A good clinical history, physical examination, electrocardiogram and x ray fluoroscopy are all essential Often auxiliary aids, such as a jugular pulse tracing, help differentiate mitral regurgitation from tricuspid insufficiency in cases in which a systolic murmur is heard just medial to the apex

Even with the greatest of care in diagnosis in the best of clinics, errors may occur Some patients with clinical diagnoses of mitral stenosis have been found at operation to have free mitral regurgitation and no stenosis Some cases diagnosed as cor pulmonale have been found at postmortem examination to be tight mitral stenosis, and others diagnosed as severe mitral regurgitation with slight mitral stenosis have turned out to be cases of tricuspid regurgitation with tight mitral stenosis

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used the term "protosystolic aortic clap," and in 1945, Froment and Coblentz[4] gave it the name of 'mesosystolic aortic vibration" It is composed of a fairly high intensity, medium frequency group of vibrations occurring 0 12 to 0 20 sec after the onset of the Q wave in the electrocardiogram and 0 08 to 0 10 sec after the initial vibrations of the first heart sound Generally exceeding the first heart sound in amplitude, it occurs after the opening of the sigmoid valves

In a study of 15 young persons who had aortic stenosis, Rheinhold and his co workers[5] found 6 of them to have an early systolic click occurring on on average of 0 18 sec after the beginning of the Q wave of the electro cardiogram The frequency of this sound was between 150 and 200 cycles per second

Since the first heart sound is described as having vibrations arising in the great vessels which constitute its usually inaudible fourth component, this early systolic click could be considered as being a delayed and exaggerated fourth part of the first sound and not as a truly new sound

Second sound

The aortic second sound may be diminished or even absent in aortic in sufficiency, or it may blend with the aortic diastolic murmur, which has its greatest intensity early in diastole In aortic stenosis the aortic second heart sound may be loud normal or even absent Frequently it is stated that the second sound is normal in subaortic stenosis and diminished in aortic valvu lar stenosis,[6] but we are not aware of any good evidence to support this statement Usually it is diminished in severe aortic stenosis, and also in moderate or even mild aortic stenosis when associated with myocardial failure from any cause Mild aortic stenosis calcific aortic stenosis or stenosis associated with systemic arterial hypertension may have a normal or even a loud second heart sound

In their study of 15 cases of aortic stenosis, Rheinhold and his associates[5] observed that in 14 the average splitting of the second heart sound was 0 02 sec compared with 0 04 sec in 25 normal persons It is possible that this narrow splitting may be due to prolonged left ventricular systole, re sulting in delay of the aortic valve closure Gray[7] reported paradoxical splitting where aortic valve closure occurs after pulmonic valve closure and the degree of splitting narrows during inspiration when pulmonic closure is also delayed

In some cases of aortic stenosis, the second heart sound may be widely split In one of our patients who had severe aortic stenosis, with a gradient of 85 mm of mercury across the aortic valve, the second sound was split by 0 06 sec

In luetic aortitis, the aortic second sound may have a rather metallic quality described as "tambour like', this may be due to calcification of the ascending aorta

13 / Aortic Valve

Like mitral valve disease, aortic valve disease may consist of stenotic or insufficiency lesions, or a combination of both When the aortic valve is involved in rheumatic fever, insufficiency always occurs first, stenosis may be a later development Luetic aortitis (see pp 350, 357) does not affect the valve cusps, and aortic insufficiency without stenosis results Congenital aortic valvular disease usually results in stenosis, either subaortic or valvular, but minor degrees of insufficiency may exist Occasionally there may be a defective medial cusp of the aortic valve or congenital fenestration of the valve leaflets, giving rise to aortic insufficiency Acquired lesions affecting the ascending arch of the aorta, such as aneurysm (either fusiform or dissecting), may result in dilatation of the aortic ring with insufficiency

The murmurs of aortic valve lesions indicate whether stenosis, insufficiency or both are present, but they are not diagnostic of the degree of malfunction and do not indicate the underlying cause

HEART SOUNDS IN AORTIC VALVE DISEASE

First sound

In diseases of the aortic valves there may be marked accentuation of the first heart sound because of (1) the increased force of left ventricular contraction, giving a hyperdynamic heart with vigorous mitral valve closure, or (2) changes in the sigmoid valves which may make their opening more audible The third component of the first heart sound sometimes has an almost cracking sound The two peaks of the first heart sound may be separated from 0 05 to 0 07 sec

Early systolic click

Following the first heart sound, there may be an additional early systolic sound which is loud and can be heard best over the aortic area This sound occurs in cases of a dilated aorta associated with either aortic stenosis or insufficiency, but more commonly with the abrupt aortic expansion in predominant aortic insufficiency

Potain [1] considered this sound to be due to an early, sudden, and limited distension of the aortic walls, and Wolferth and Margolies [2] observed it to be synchronous *with the carotid upstroke* In 1941, Lian, Minot, and Welti [3]

used the term "protosystolic aortic clap," and in 1945, Froment and Coblentz[4] gave it the name of 'meso systolic aortic vibration" It is composed of a fairly high intensity, medium frequency group of vibrations occurring 0 12 to 0 20 sec after the onset of the Q wave in the electrocardiogram and 0 08 to 0 10 sec after the initial vibrations of the first heart sound Generally exceeding the first heart sound in amplitude, it occurs after the opening of the sigmoid valves

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AORTIC INSUFFICIENCY

Etiology and diagnosis

Aortic insufficiency may be caused by a wide variety of diseases or conditions rheumatic fever (Fig 81) (see p 237), syphilis, bacterial endocarditis (Figs 82 and 83), traumatic rupture of the valves, calcific arteriosclerotic aortic valve deformity, dissecting aneurysm of the ascending aorta with distension of the valve ring, usually occurring in patients with hypertension (from any cause) or cystic medial necrosis of the aorta,

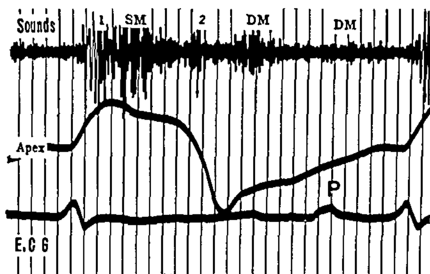


Fig 81 (2 R I S Log) Systolic and diastolic murmurs in rheumatic heart disease Note the normal first sound (1) 0 09 sec after the onset of the QRS complex the somewhat diamond shaped systolic murmur (SM) of medium frequency and high intensity the normal second sound (2) and the long diastolic murmur (DM) with a short crescendo and a long decrescendo phase

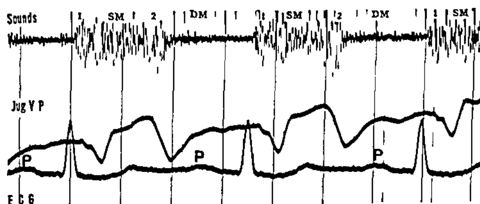


Fig 82 (2 R I S Log) Aortic systolic and diastolic murmurs in rheumatic heart disease with subacute bacterial endocarditis and combined mitral and aortic lesions Note the normal intensity first sound (1) 0 07 sec after the onset of the QRS complex the normal second sound (2) the systolic murmur (SM) of medium frequency and moderate intensity with a tendency toward a diamond shape and the low frequency moderate intensity diastolic murmur (DM) continuing throughout diastole

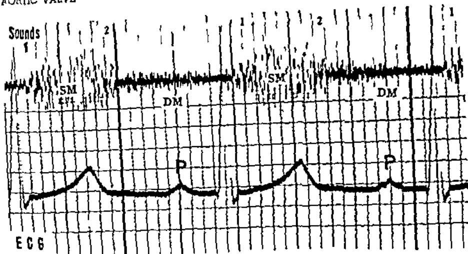


Fig 83 (2 R.J S Log) Diamond shaped systolic murmur and a diastolic murmur over the base of the heart of a patient with aortic regurgitation following an attack of bacterial endocarditis involving the aortic valve Note the low frequency low intensity first sound (1) 0 09 sec after the onset of the Q wave the second sound (2) almost completely obscured by the murmurs the diamond shaped holosystolic murmur (SM) of high frequency and high intensity with the apex of the diamond in mid systole and the decrescendo diastolic murmur (DM) lasting throughout diastole

rheumatoid arthritis cardiac dilatation with severe anemia (Fig 84), hypertension, senile ectasia of the aorta "eversion" of a valve cusp (Fig 85), and in some cases by aortic valve surgery for the correction of aortic stenosis Congenital lesions causing aortic insufficiency are fixed opening of the valve in aortic stenosis (Fig 86), bicuspid aortic valve, fenestration

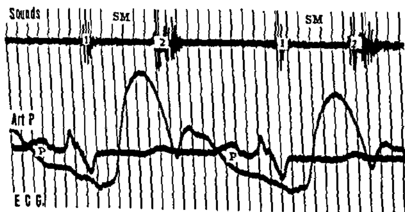


Fig 84 (2 R 1 S Log) Decrescendo second sound with a few after vibrations but not a clear cut diastolic murmur in cirrhosis of the liver and vitamin B deficiency Note the first heart sound (1) of normal intensity 0 11 sec after the Q wave of the electrocardiogram the few vibrations in systole (SM) not sufficient to classify as a true systolic murmur and a second sound (2) split by 0 03 sec Following the second component of the second sound is a series of decrescendo vibrations which probably represents a slight short diastolic insufficiency blow

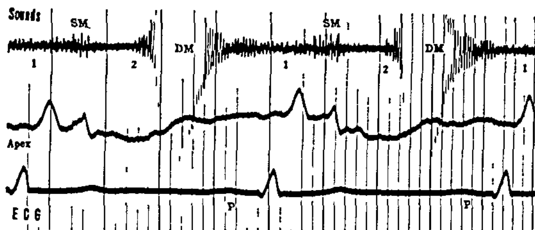


Fig 85 (2 R I S Log and apex cardiogram) Musical diastolic murmur of aortic insufficiency with an everted cusp Note the low intensity first heart sound (1) the crescendo decrescendo systolic murmur (SM) of low intensity and medium frequency the low intensity second sound (2) and the crescendo decrescendo diastolic murmur (DM) of extremely high intensity and high frequency The crescendic phase is short and is followed by a long decrescendic phase

of the valve leaflets, defective medial cusp with ventricular septal defect (Fig 87), coarctation of the aorta with marked hypertension, and Marfan's syndrome (Fig 88)

Murmurs

The murmur of aortic insufficiency varies in intensity, duration, and character It may be short, or it may continue throughout diastole and be terminated by the presystolic crescendo Austin Flint murmur (Fig 89) The intensity may be slight, and the murmur may be blowing in character, so that its detection is difficult, or it may be exceedingly loud and of such

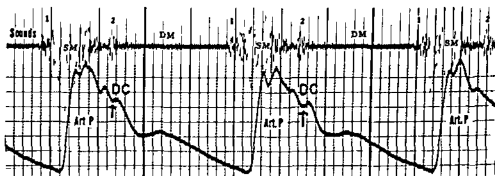


Fig 86 (2 R I S Log with arterial pulse curve) Correlation between the dicrotic notch and the aortic valve closure in a young girl with aortic valve disease probably associated with subendocardial sclerosis Note the normal first sound (1) the diamond shaped systolic murmur (SM) with the peak of the diamond in the first third of systole the normal second sound (2) the dicrotic notch (DC) of the arterial pulse indicating the time of aortic valve closure the bifid peak of the brachial pulse wave and the high frequency low intensity diastolic murmur (DM) The upstroke time of the brachial artery tracing equals 0 10 sec

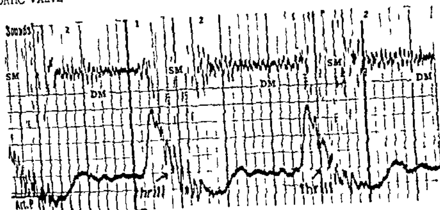


Fig 87 (2 R I S Steth) Carot d thrill in a pat ent w th a ventr cular septal defect and *aortic regu gitation* caused by a defect ve med al cusp of the aort c val e Note the normal first (1) and second (2) sounds the systol c mu mur (SM) which is d amond shaped and of h gh *intens ty and th long d astol c murmur* (DM) The arter al pulse shows h gh intensity systolic vibrat o s wh ch are synchronous w th the systol c murmur *These v brations represent the systol c thr ll*

roughness in quality that at first it may be mistaken for a systolic murmur Usually it is diminuendo, as pointed out by Fauvel[8] in 1843, although some murmurs are shown by phonocardiography to have a short early crescendo phase followed by a long decrescendo phase Transmission of the murmur to the apex is fairly common

In order to detect an aortic diastolic murmur, it is essential to have the patient hold his breath in moderate expiration The breath sounds often have a quality similar to that of the aortic diastolic murmur and may mask it completely unless respiration is halted

Because of their high frequency components, aortic diastolic murmurs can best be detected with the diaphragm chest piece of the stethoscope

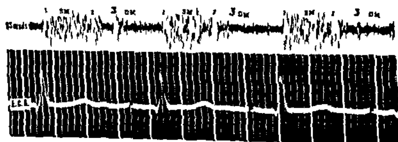


Fig 88 (2 R I S Log) To and fro murmur c used by trem ndous d latat on of the ascend ing aorta a d the aort c val e *r ng n a patient ha ng Marfan s* syndrome with cyst c de ge erat on of the aorta Note th normal first sound (1) spl t by 0 03 sec and occurring 0 08 sec aft th onset of the QRS *comple* the normal second sound (2) the somewhat d amond shap d systol c murm r (SM) of *med m* frequency and moderate intensity the th d sound (3) *ary g in inte sity* from cycle to cycle a d the diastol c murmur (DM) of med m freq ency and low to moderate tens ty last ng throughout diastole

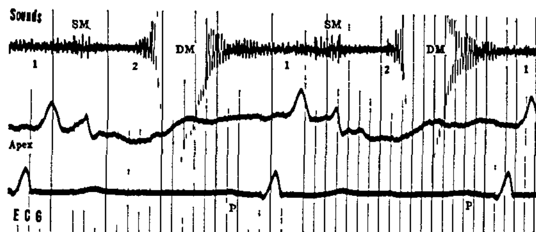


Fig 85 (2 R I S Log and apex cardiogram) Musical diastolic murmur of aortic insufficiency with an everted cusp Note the low intensity first heart sound (1) the crescendo decrescendo systolic murmur (SM) of low intensity and medium frequency the low intensity second sound (2) and the crescendo decrescendo diastolic murmur (DM) of extremely high intensity and high frequency The crescendic phase is short and is followed by a long decrescendic phase

of the valve leaflets, defective medial cusp with ventricular septal defect (Fig 87), coarctation of the aorta with marked hypertension, and Marfan's syndrome (Fig 88)

Murmurs

The murmur of aortic insufficiency varies in intensity, duration, and character It may be short, or it may continue throughout diastole and be terminated by the presystolic crescendo Austin Flint murmur (Fig 89) The intensity may be slight, and the murmur may be blowing in character, so that its detection is difficult, or it may be exceedingly loud and of such

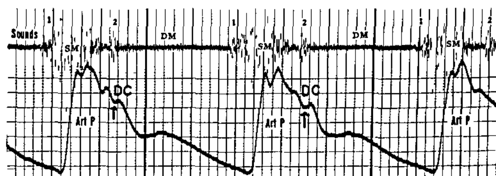


Fig 86 (2 R I S Log with arterial pulse curve) Correlation between the dicrotic notch and the aortic valve closure in a young girl with aortic valve disease probably associated with subendocardial sclerosis Note the normal first sound (1) the diamond shaped systolic murmur (SM) with the peak of the diamond in the first third of systole the normal second sound (2) the dicrotic notch (DC) of the arterial pulse indicating the time of aortic valve closure the bifid peak of the brachial pulse wave and the high frequency low intensity diastolic murmur (DM) The upstroke time of the brachial artery tracing equals 0 10 sec

of the other cusps resulted in transmission to the apex In most cases of aortic insufficiency, the insufficiency is not confined to one cusp alone, and occurs through the central valve aperture Kerr and Palmer,[14] in their review of this subject, emphasized the paucity of evidence on which the Foster rule is based, and numerous case reports have failed to prove its clinical consistency

Aortic insufficiency and pulmonary insufficiency

One of the most difficult diagnoses in medicine is the differentiation of the murmur which occurs in mild degrees of aortic insufficiency from the Graham Steell murmur of pulmonary insufficiency In mild cases of aortic insufficiency, there are no helpful peripheral signs (Fig 90), and, whereas in some cases of pulmonary insufficiency the pulmonic second sound may be markedly accentuated, this is not always so

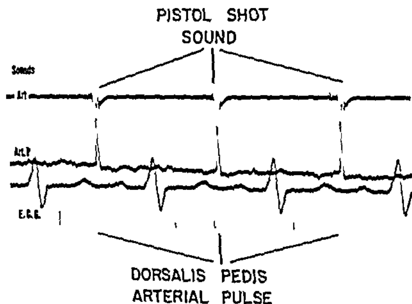


Fig 90 (Simultaneous phonocardiogram and arterial pulse tracing over the dorsalis pedis artery together with an electrocardiogram) Pistol shot sound over the dorsalis pedis artery in a 12 year old white boy with rheumatic heart disease and free aortic regurgitation Note the pistol shot sound 0 38 sec after the onset of the QRS complex and the sudden rise and fall of the arterial pulse wave

One helpful suggestion, in acquired heart disease particularly, is not to make a diagnosis of pulmonary insufficiency unless there is a palpable second sound at the pulmonic area Another is to exercise the patient, during exercise the Graham Steell murmur is said to get louder because of the rise in pulmonary artery pressure However, we have not found this procedure to be reliable, and there are some cases of "idiopathic" pulmonary insufficiency in which the pulmonic second sound is normal, in congenital hypo

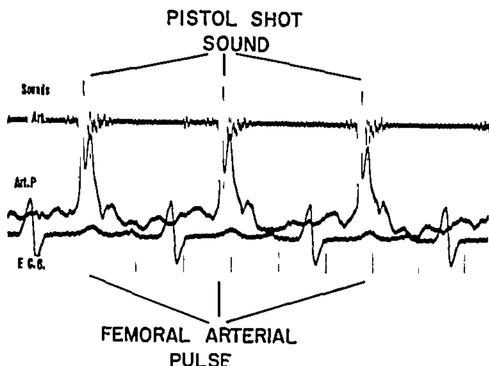


Fig 89 (Phonocardiogram over femoral artery with simultaneous femoral arterial pulse tracing and electrocardiogram) Pistol shot sound over the femoral artery in a 12 year old white boy with rheumatic heart disease and free aortic regurgitation Note the loud pistol shot sound 0 26 sec after the onset of the QRS complex and the sudden rise and slightly more gradual fall of the arterial pulse wave

The diaphragm tends to screen out the lower frequency vibrations, making the higher frequency components appear stronger

In the presence of a ruptured or a so called "everted cusp," and with calcification of the aortic valve, the murmur may be musical Loud musical *murmurs are likely to be evanescent, the reason for this is not known*

Comparatively loud aortic systolic murmurs may be heard in the presence of pure aortic insufficiency The probable reason for this is the large increase in stroke volume, with a greatly increased flow across the aortic valve, causing relative aortic stenosis Another possible reason for this murmur is the formation of eddy currents in the dilated proximal portion of the aorta These murmurs are maximal in early or mid systole, and are transmitted to the suprasternal notch and along the great vessels of the neck, more especially to the right than to the left

FOSTER'S *MURMUR AND RULE* *Between 1866 and 1873,* Foster [9 10 11] described several cases of ruptured aortic valve cusps in which he tried to correlate the direction of transmission of the murmur with the particular cusp affected He believed that rupture of the posterior or noncoronary cusp resulted in transmission of the murmur to the apex and that rupture of either of the coronary cusps resulted in transmission to the ensiform process On the other hand, Herrmann,[1 13] in studies on animals, found that rupture of the posterior cusp resulted in the Flint type of murmur, whereas rupture

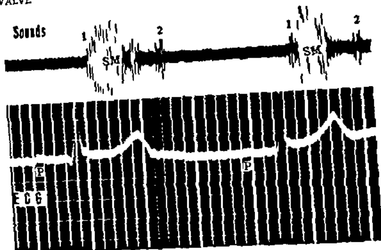


Fig 91 (2 R I S Log) Early diamond shaped systolic murmur in a young boy with congenital aortic stenosis Note the first sound (1) of low intensity occurring 0 08 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) occupying the first three fourths of systole (there is an early crescendic phase followed by a longer decrescendic phase) and the second sound (2) of normal intensity and split by 0 02 sec



Fig 92 (2 R I S Log) The effect of a ventricular ectopic beat on the heart sounds and murmurs in postoperative aortic stenosis and insufficiency Note the effect of the ventricular ectopic beat on the onset of the first heart sound (1) This sound normally begins 0 06 sec after the onset of the QRS complex but it begins 0 17 sec after the onset of the QRS with the ectopic beat The systolic murmur (SM) is greatly reduced in intensity with the ectopic beat and the diastolic murmur cannot be seen clearly The second heart sound which was normally split (2 2) cannot be identified following the ectopic beat

One reason why aortic stenosis has been missed frequently in the past is that physicians, while diligent in listening at the cardiac apex, often are less conscientious in their auscultation at the base of the heart The auscultatory criteria usually accepted for diagnosing aortic stenosis are (1) a grade 2 (or greater) 'stenotic' systolic murmur at the second right interspace, conducting well to the suprasternal notch and the back, (2) an associated systolic thrill, and (3) a normal or diminished aortic second sound

These criteria are suitable for the uncomplicated cases of valvular aortic stenosis, but they are not quite so applicable in aortic stenosis with additional lesions (such as mitral stenosis or myocardial fibrosis), in cases of congestive failure, or in primary myocardial disease, such as subendocardial sclerosis

plasia of the pulmonary valves, the second sound may be diminished or absent

If additional lesions, such as tight mitral stenosis, are known to be present, and if the pulmonic second sound is accentuated, pulmonary insufficiency may well be suspected, but to prove this is exceedingly difficult, even after cardiac catheterization and a careful study of the ventricular and pulmonary artery pressure curves The differential diagnosis may not be possible on clinical grounds alone

Postmortem inspection of the aortic valves in cases of severe calcific aortic stenosis with rigid valves, shows a fixed aperture between the valve cusps It seems obvious that aortic regurgitation must be present in these instances, and this theory is borne out by the presence of an aortic diastolic murmur in approximately 20 per cent of the cases in our series It is possible that aortic regurgitation may not be present in the early stages of the disease but may become marked as the valve deformity increases Failure of the regurgitant jet to produce an audible murmur is presumably due mainly to the small regurgitant flow secondary to the cone shaped deformity of the valve with its apex "upstream "[1]

DISSECTING ANEURYSM

In the differential diagnosis of aortic systolic and diastolic murmurs, one must consider dissecting aneurysm of the aorta Classically regarded as occurring in older persons with hypertension, the onset is said to be accompanied by a severe tearing pain in the chest It is not generally appreciated that dissection may be painless in 25 per cent of the patients It has been reported in a fourteen month old patient, approximately 10 to 20 per cent of the patients are less than forty years of age, and previous hyptertension is not a sine qua non It may occur spontaneously in pregnant young women

The diagnosis of dissecting aneurysm of the aorta can be made easily when the sudden onset of the classical signs is followed by a prolonged course of congestive heart failure with cardiomegaly, tachycardia, and tachypnea Aortic insufficiency is secondary to the aneurysmal dilatation of the aorta which accompanies the aortic dissection

AORTIC STENOSIS

ETIOLOGY AND DIAGNOSIS

Congenital aortic stenosis is fairly common (Figs 91 and 92) For many years there has been great controversy as to whether aortic stenosis with a calcified valve in adults is secondary to a rheumatic (Fig 93) or an arteriosclerotic process Since aortic stenosis occurs in children, and since the majority of these patients survive their childhood, it is reasonable to suppose that a certain number of cases of aortic stenosis in adults are congenital, although some may be rheumatic or arteriosclerotic in origin

Since the systolic murmur does not begin until after the isometric contraction of the ventricle, mitral valve closure is not affected, and the first heart sound can be heard clearly The murmur has its greatest intensity during the phase of maximal ejection from the ventricle, and this occurs early in systole

The murmur is typically loud and rough, systolic in timing and most marked early in systole It is heard best at the aortic area and is well conducted to the neck, particularly to the right side, and occasionally also over the upper part of the precordium Frequently, it is heard clearly along the left sternal border, and sometimes also at the cardiac apex Lian and Geismar[21] noted that the maximal site for dorsal propagation was the medial aspect of the right supraspinous fossa The murmur terminates before the aortic second sound, which may be normal or diminished in intensity, in those cases associated with aortic regurgitation, the second aortic sound may even be completely absent

The murmur of aortic stenosis is always rough even if it is low in intensity In the thick chests of muscular or fat persons or in emphysematous patients it may be difficult to hear

In some patients a high pitched murmur, variably described as squeaking or musical in character, may be heard at the apex This murmur was described by Stokes in 1854 If a high pitched, squeaking or musical murmur is heard at the apex in elderly persons, a careful search should be made at the base to try *to identify* a rough aortic systolic murmur

It must be realized that since aortic stenosis in its early stages may have few, if any clinical signs the lesser degrees of aortic stenosis may not present the classical murmur and thrill Any roughness of a basal systolic murmur should arouse *suspicion*, and the patient should be followed over a period of years to see if the murmur develops the characteristics of typical aortic stenosis

It is not necessary to find a small pulse or a slow upstroke of the pulse wave before making the diagnosis of aortic stenosis, nor is it necessary to detect a systolic thrill All these factors are merely corroborating evidence of its existence being useful when present but not excluding the diagnosis if absent

TRIGONOIDATION ' In 1937 Chisholm [3] introduced the term 'trigonoidation' to explain the presence of an aortic systolic murmur with dilatation of the proximal portion of the aorta but without organic narrowing of the aortic valve He pointed out that since the aortic valves were held *at fixed* points by their insertions they could not open to the full size of the dilated aorta therefore the three cusps would protrude into the aortic orifice in the form of a triangle thus narrowing the aortic *orifice* These valves could then vibrate *freely* in the column of blood creating a murmur The same condition may exist at the pulmonic valve



Fig 93 (2 R I S Log) Aortic systolic and diastolic murmurs in a patient with rheumatic heart disease atrial fibrillation and aortic stenosis and insufficiency Note the normal first heart sound (1) 0 08 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) of very high intensity and medium frequency with the apex of the diamond just before mid systole the second heart sound (2 2) split by 0 04 sec and the long slightly decrescendo diastolic murmur (DM) of moderate intensity commencing with the second part of the split and continuing throughout diastole

Probably the earliest sign of aortic stenosis is a slight, rough basal systolic murmur, heard best over the second right interspace, but this alone is not sufficient evidence for a definite diagnosis, and one must await the development of more classical signs

In children, aortic stenosis may commence as a rather rough systolic murmur heard at the third left intercostal space, only after some years does the typical murmur and its accompanying thrill at the second right interspace become evident (Taussig[16] reported 2 such cases in 1955) Levine[17] pointed out that in many patients, originally regarded as having benign, inconsequential systolic murmurs when there were no symptoms or other evidence of heart disease, a loud systolic murmur and calcific aortic stenosis developed ten to twenty years later Wiggers[18] pointed out that the aortic orifice must be reduced to something less than one fourth of its normal size before significant obstruction to left ventricular outflow occurs

In most cases, the pulse is regular but there may be atrial fibrillation In moderate to severe cases there is an associated aortic systolic thrill or, rarely, a thrill confined to the mitral area

Murmurs

Evans,[19] in 1947, pointed out that the systolic murmur of aortic stenosis begins immediately after the first heart sound in some patients and after a slight delay in others In 19 of 20 cases studied by Leatham,[20] the summit of intensity of the systolic murmur occurred in mid systole, but we have found the summit to occur more often earlier in systole

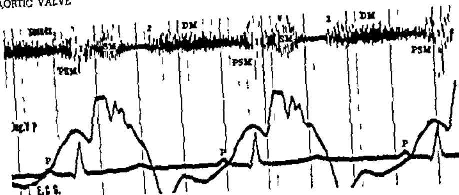


Fig 95 (Apex Log) A presystol c crescend c murmur in a 56 year old man with syph l t c aort t s and aort c insuff ciency Note the f rst sound (1) of normal intens ty occurr ng 0 08 sec after the onset of the QRS complex the d amo d shaped systol c murm r (SM) of med um frequency and moderate intens ty occupy g the f rst two th rds of systole a second sound (2) of very low intensity and a d astol c murmur (DM) rather h gh in frequency and of moder te tensity terminat ng in a true p esystol c murmur (PSM)

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AORTIC STENOSIS IN THE PRESENCE OF SEVERE MITRAL STENOSIS OF MYOCARDIAL FAILURE

Severe degrees of aortic stenosis may be completely masked by the red tion in cardiac output which accompanies marked degrees of mitral sten or myocardial failure Some patients with mitral stenosis have underg surgery only to die postoperatively because of previously unsuspected vere aortic stenosis

THE SYSTOLIC THRILL IN AORTIC STENOSIS

A systolic thrill in the aortic area, at the suprasternal notch, and over great vessels of the neck frequently accompanies the systolic murmur aortic stenosis In milder cases with relatively low intensity murmurs thrill may not be palpable, its presence is a reflection of the energy tra mission of the murmur, and is in no way indicative of the severity of lesion

COMBINED AORTIC STENOSIS AND AORTIC INSUFFICIENCY

If both systolic and diastolic aortic murmurs are present, they may caused either by pure aortic insufficiency or by combined aortic insufficien and stenosis If free aortic regurgitation is present, as shown by periphe signs, it is reasonable to assume that the degree of aortic stenosis probab is not significant Again, if mild aortic regurgitation is present, as shown the peripheral pulses (Figs 94 and 95) and blood pressure, and yet card megaly with marked left ventricular hypertrophy, and perhaps angina, present, then stenosis is likely to be the dominant lesion

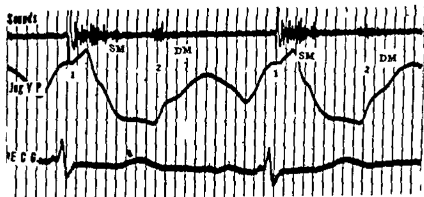


Fig 94 (2 R I S Log) A diminished second sound and minimal aortic insufficiency in 68 year old man with syphilitic aortitis and dilatation of the ascending aorta Results Hinton test were positive Note the first heart sound (1) of normal intensity occurring 0 C sec after the onset of the QRS complex the decrescendo systolic murmur (SM) of mediu frequency and moderate intensity the greatly diminished second heart sound (2) and th early diastolic murmur (DM) of very low intensity commencing with the second soun



Fig 95 (Apex Log) A presystol c cresce d c murmur in a 56 year old man with syphil t c aort tis and aort c insuff c ency Note the first sound (1) of normal intens ty occurri g 0 08 sec after the onset of the QRS complex the d amond shaped systol c murmur (SM) of med um frequency and moderate intens ty occupy ng the first two th rds of systole a second sound (2) of very low i tens ty and a d astol c murmur (DM) rather h gh in f equency and of moderate inte s ty term nat ng in a t ue p esystol c murmur (PSM)

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14 / Pulmonic Valve

INSIGNIFICANT PULMONIC SYSTOLIC MURMURS

The great majority of pulmonic systolic murmurs are classified as "not significant." These murmurs are soft and blowing in quality and are grade 1 to 2 in intensity. They can be heard in most infants, children, and young adults, and frequently they can be detected in older persons. They are not associated with any apparent heart disease. In all probability, they are due to turbulence resulting from blood flow across the pulmonic valve and are heard easily because of the anterior position of the pulmonic valve in the chest.

SIGNIFICANT PULMONIC SYSTOLIC MURMURS

"Significant" systolic murmurs of the pulmonic valve are most frequently associated with congenital heart lesions, with organic pulmonic stenosis, with increased blood flow through the pulmonic valve (secondary to left to right shunts at the atrial or ventricular levels) or even with peripheral arteriovenous aneurysms. The systolic murmur of valvular pulmonic stenosis will be discussed in detail in the section on congenital heart disease (pp. 297-299). It is sufficient to state that this murmur (1) is heard at or shortly after birth, (2) is loudest at the second left intercostal space, (3) is rough and "stenotic" in quality, (4) radiates well to the upper part of the back, the suprasternal notch and also to the neck, (5) is usually grade 3 to 4 in intensity, (6) is nearly always accompanied by a systolic thrill, (7) is diamond shaped in the phonocardiogram, and (8) *in the more severe cases* is terminated by a diminished pulmonary valve closure sound.

Rarely pulmonic stenosis may be acquired; if so, it may occur as an isolated lesion associated with carcinoid tumors (Bjork's syndrome) or in conjunction with multivalvular rheumatic heart disease, in which case its significance is overshadowed by that of the other valve lesions.

SOUNDS AND MURMURS OF PULMONARY VALVE INSUFFICIENCY

First heart sound

The first heart sound may be normal in intensity or may be markedly accentuated, especially with associated mitral stenosis or with the hyper

dynamic heart of some congenital lesions, it is loudest at the apex or lower left sternal border and is often followed by an early systolic ejection sound (systolic click) occurring after the opening of the semilunar valves and heard best at the second and third left interspace (Fig 96)

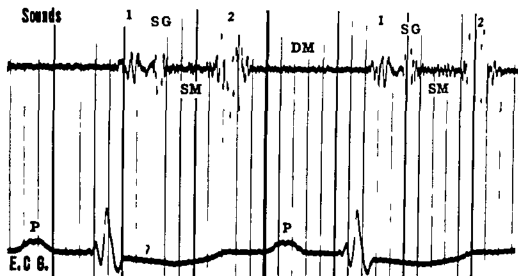


Fig 96 (2 L I S Log and electrocardiogram) Early systolic click (SG) in an asymptomatic patient with idiopathic dilatation of the pulmonary artery The first sound (1) is of moderately low intensity and occurs 0 08 sec after the onset of the QRS complex The early systolic click (SG) occurs 0 16 sec after the onset of the QRS complex Note the systolic murmur (SM) of medium frequency and low intensity the prominent second sound (2) and the artefactual diastolic vibrations (DM)

Pulmonic second sound

In most cases of pulmonary insufficiency associated with hypertension of the pulmonary artery, pulmonic valve closure is accentuated in intensity—often markedly It is the loudness of the pulmonic second sound which leads one to listen carefully for the insufficiency blow

In idiopathic pulmonary insufficiency, the second sound may be normal, whereas after the surgical correction of valvular pulmonic stenosis, it is normal or diminished

Diastolic murmurs

The pulmonary regurgitant murmur, first described by Graham Steell,[1] is one of the most difficult murmurs to identify in the field of auscultation Often the patient with a pulmonary regurgitant murmur has a history of rheumatic fever, and the possibility of confusing this murmur with that caused by aortic insufficiency arises

There are some points which help in the identification of a Graham Steell murmur, however, none of these is positive proof that aortic regurgitation has been excluded

1 The murmur is localized to the second and third left intercostal spaces
2 There is associated pulmonary hypertension as shown by accentuation of the pulmonary second sound
3 The peripheral signs of aortic insufficiency such as wide pulse pressure capillary pulsation pistol shot femorals or bounding peripheral pulses are absent
4 There is no clinical or serologic evidence of syphilis

CHARACTER OF THE MURMUR Usually the murmur of pulmonary insufficiency is soft, high pitched, decrescendo, and blowing, starting with the closure of the pulmonic valve and ending in early or mid diastole Since the murmur usually is associated with pulmonary artery hypertension, it may be preceded by an accentuated pulmonic second sound, which may be palpable The site of maximum intensity of the murmur and of the pulmonic valve closure should be the same, namely, the first, second or third left interspace at the sternal border The Graham Steell murmur is not always a short low intensity murmur, occasionally it is not only loud and easily heard, but it may continue throughout diastole and have presystolic accentuation, like the Austin Flint murmur of aortic regurgitation

ASSOCIATED WITH HYPERTENSION OF THE PULMONARY ARTERY Pulmonary vascular obstruction always causes pulmonary artery hypertension As the degree of hypertension increases, the likelihood of pulmonary insufficiency increases and the characteristic murmur may become audible

Congenital murmurs result from primary pulmonary vascular obstruction (Fig 97) Acquired ones may result from (1) severe mitral stenosis, (2)

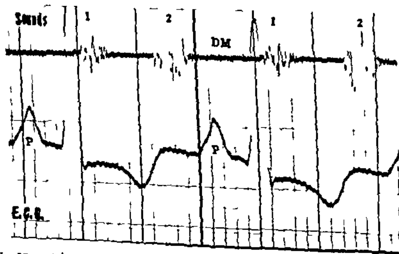


Fig 97 (2 L I S Log) Short pulmonic insufficiency murmur in a 12 10/12 year old white girl with primary pulmonary vascular obstruction and congestive heart failure Pressures pulmonary artery 134/54 mm Hg mean = 80 mm Hg right ventricular 134/3/18 mm Hg right atrial mean = 10 mm Hg a waves = 17 mm brachial artery 83/53 mm Hg Note the first sound (1) of normal intensity commencing 0 08 sec after the onset of the Q wave and split by 0 03 sec The second sound (2) which is of high frequency and moderate intensity has a decrescendo termination probably representing a slight Graham Steell murmur

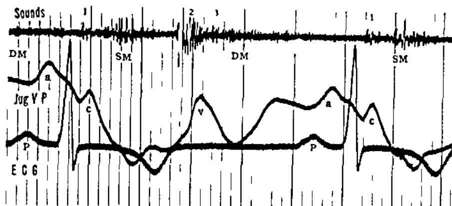


Fig 98 (2 L I S Log) Diastolic murmur of pulmonary insufficiency in a patient with pulmonary vascular obstruction and pulmonary artery hypertension secondary to multiple pulmonary emboli Note the first sound (1) of low intensity the systolic murmur (SM) of moderate intensity and medium frequency the very loud second sound (2) a low intensity third sound (3) and a diastolic murmur (DM) starting with the second sound and continuing into diastole in decrescendo fashion

chronic obstructive lung disease, (3) obstructive vascular disease of the lungs (such as multiple small pulmonary emboli or multiple carcinoma cell metastases (Fig 98), or (4) pulmonary vascular obstruction associated with large left to right shunts at the atrial (Figs 99 and 100), ventricular (Fig 101), or pulmonary artery (Fig 102) levels This latter group includes shunts created surgically

Although the murmur of pulmonary artery hypertension was first described by Graham Steell in a patient with mitral stenosis, it is obvious that a similar murmur could result from pulmonary artery hypertension, what

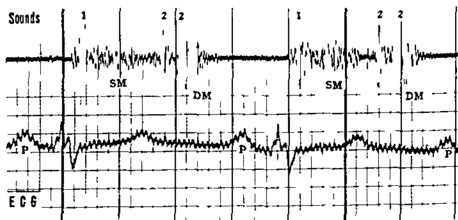


Fig 99 (2 L I S Log) Pulmonic valve insufficiency as shown by Graham Steell murmur in patient with atrial septal defect and pulmonary artery hypertension Note the normal first heart sound (1) 0 10 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) of medium frequency and moderate intensity the widely split second sound (2 2) split by 0 07 sec with an accentuated second component and the early decrescendo diastolic murmur (DM) of high frequency starting with pulmonic valve closure and lasting throughout the first quarter of diastole

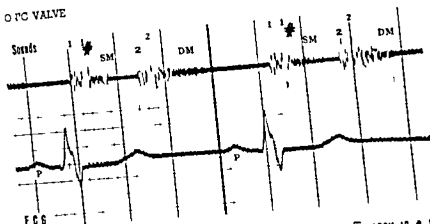


Fig 100 (2 L.I S Log) Graham Steell murmur of pulmonary insuff c ency in a patient w th an at al septal defect and pulmonary a tery hypertens on Note the normal f rst heart so d (1) 0 08 sec after the onset of the QRS complex the early systol c cl ck (1#) 0 14 s c afte the onset of the QRS complex the early decrescendo systol c murmur (SM) the well spl t (0 04 sec) second sound (2 2) w th accentuat on of the second component or pulmona y closu e and the decresce do d astol c murmur (DM) of h gh frequency and moderate ntens ty commenc ng with pulmon c valve closure and continu ng throughout the g eater part of d astol

ever the cause The murmur is cau ed by the regurgitation of blood from the pulmonary artery through the pulmonic valve into the right ventricle, but the loudness of the murmur is not necessarily indicative of the degree of regurgitation

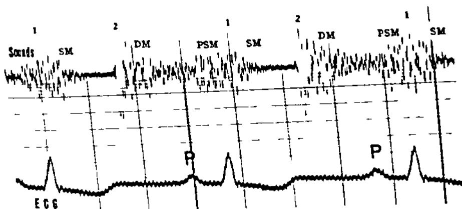


Fig 101 (2 L.I S Log) D astol c murmur of pulmo c ins ff cie cy w th presystol c cre scendo assoc ated with p lmonary hyperte s o in pat e t w th a lo g stand g left to right shunt at th ve t cul r l vel Note the f st sou d (1) of normal intensity occurring probably 0 08 se afte the onset of the QRS c mpl x (altho gh n the f st and last complexes the ma m l b at o s seem to be s multaneous with the peak of the R wave) Note also the short dec esce d c systol c mu mur (SM) of low intens ty the loud seco d sound (2) and th d stol c m mu (DM) of h gh intens ty and med um to h gh freq ency commenc ng w th the s ond sou d a d te m nat ng n a p esystol c crescend c murmur (PSM) This type of p systol c cr ndo occu ng w th th m rmur of pulmonary nsuff c ency is comparable to the A st Fl t mu m r of ort c insuff cie cy

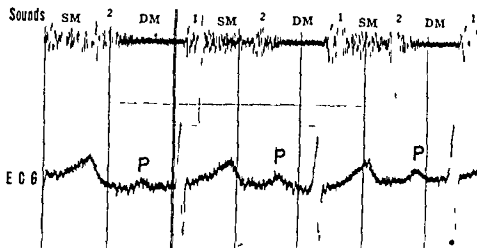


Fig 102 (2 L I S Log) Murmurs heard at the second left interspace in a patient with a patent ductus arteriosus and pulmonary artery hypertension The loud first heart sound (1) is apparently split by 0 04 sec The first part of the split follows the onset of the QRS complex by 0 05 sec The second part of the split is much louder follows the onset of the QRS complex by 0 09 sec and undoubtedly represents an early systolic click Note the decrescendo systolic murmur (SM) of moderate intensity and medium frequency the second sound (2) of variable intensity and the decrescendo diastolic murmur (DM) of low intensity and high frequency starting with the second sound and continuing throughout the first half of diastole It is impossible to be certain either clinically or phonocardiographically whether this murmur is a pulmonary insufficiency blow or the diastolic component of a ductus murmur although the high frequency suggests an insufficiency blow

NOT ASSOCIATED WITH HYPERTENSION OF THE PULMONARY ARTERY Murmurs of pulmonary insufficiency not associated with hypertension of the pulmonary artery may also be either congenital or acquired Congenital ones (Fig 103) result from some anomaly of the pulmonic valve cusps or the pulmonic valve ring This is not common, it may represent an isolated lesion,

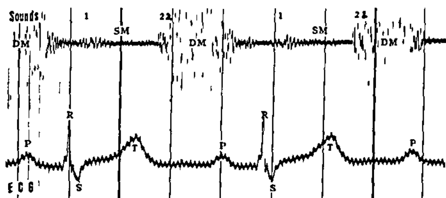


Fig 103 (2 L I S Log) Loud diamond shaped diastolic murmur of pulmonic insufficiency secondary to idiopathic pulmonary insufficiency in an asymptomatic patient Note the first heart sound (1) of low intensity 0 06 sec after the onset of the QRS complex the minimal systolic murmur (SM) the normal second sound (2 2) split by 0 03 sec and the extremely high intensity medium frequency *crescendo decrescendo diastolic murmur (DM)* commencing with pulmonic closure and continuing throughout the first three quarters of diastole

and in the few cases of this type which we have observed, the murmur has not been associated with any symptoms or other signs of congenital heart disease Congenital anomalies of the pulmonic valves may also be associated with ventricular septal defect, occasionally in association with infundibular pulmonic stenosis The acquired murmurs result from surgical distortion of the pulmonic valve after attempted correction of congenital pulmonic stenosis (Fig 104), either from splitting of the pulmonic valve itself or secondary to the insertion of a prosthesis across the pulmonary valve ring

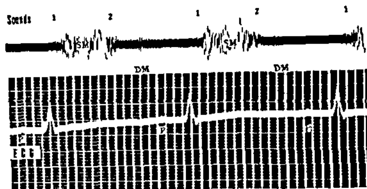


Fig 104 (2 L I S L g) Short diastolic murmur of pulmonic insufficiency following Brock valvotomy for pulmonic stenosis The first sound (1) is of low intensity and occurs 0 07 sec after the onset of the QRS complex Note the crescendo decrescendo systolic murmur (SM) of moderately high frequency and intensity the second sound (2) of low intensity and the diastolic murmur (DM) of low intensity and medium frequency occupying the first half of diastole

PULMONARY INSUFFICIENCY ASSOCIATED WITH MITRAL STENOSIS The diagnosis of a Graham Steell murmur is often overlooked in cases of severe mitral stenosis not because it is not suspected, but because aortic regurgitation is often associated with rheumatic mitral disease, and the difficulties in excluding this condition are well known

With the great increase in mitral valve surgery in recent years, it has become commonplace to hear a diastolic murmur at the second and third left interspaces preoperatively, and to be unable to decide whether it is aortic or pulmonic in origin Complete disappearance of the murmur after successful splitting of the mitral valve suggests pulmonic origin, whereas an increase in the murmur postoperatively suggests that it is due to aortic regurgitation, which has become more marked with the increase in cardiac output after relief of the severe mitral obstruction

If the murmur neither increases nor decreases, the point cannot be proved one way or the other this is especially so if the pulmonary hypertension persists after surgery

CONCLUSION The murmur of pulmonary insufficiency may accompany

many different forms of underlying heart disease, but in the majority of cases there is an associated increase in pulmonary artery pressure

REFERENCE

1 Steell, G The murmur of high pressure in the pulmonary artery M Chron 9 182 1888

15 / Tricuspid Valve

Tricuspid valve disease may be congenital or acquired, it may cause regurgitation or stenosis or both The accompanying murmurs may be due to structural changes in the valve cusps or the valve ring or they may be due to increased blood flow through a relatively normal valve

Acquired tricuspid disease as an isolated valve lesion is rare Except for a few cases associated with disseminated lupus erythematosis or carcinoid tumors the disease is practically always accompanied by concomitant mitral lesions and sometimes by aortic valve disease also The close anatomic relationship between the tricuspid and mitral valves makes the separation of their murmurs extremely difficult Although the diagnosis of acquired tricuspid valve disease is being made with more and more accuracy, nevertheless, difficulties are still frequently encountered in its clinical recognition The electrocardiogram and the roentgenogram are of little help in the diagnosis

The accuracy of the antemortem diagnosis of tricuspid valve disease does not compare favorably with that of aortic or mitral valve disease Frequently, tricuspid valve disease is found at postmortem examination when it had not been suspected during life Smith and Levine,[1] studying the results of 340 postmortem examinations of persons with rheumatic heart disease, including 227 with mitral stenosis, found 32 cases of tricuspid stenosis It was severe in 11 cases, moderate in 11 and slight in 10 mitral stenosis was present also in all 32 cases In an additional 60 cases, there was organic tricuspid valve damage which was usually severe enough to cause functional tricuspid insufficiency In other words, some form of organic tricuspid valve involvement was present in 27 per cent of the 340 cases of chronic rheumatic heart disease Of the 32 persons who had tricuspid stenosis, the average age at death was 34 3 years There were 9 males and 23 females none of the males had marked tricuspid stenosis

Because of the difficulties in making an accurate diagnosis of tricuspid valve disease on the basis of auscultation other points in the physical examination will be discussed briefly in this chapter

TRICUSPID INSUFFICIENCY

Congenital anomalies

Tricuspid insufficiency may occur with certain congenital anomalies involving the tricuspid valve Among these are Ebstein's anomaly and defects

in the upper part of the ventricular septum at the origin of the medial cusp of the tricuspid valve Examples of the latter may be found with atrioventricularis communis defects

Other congenital lesions causing tricuspid insufficiency do not involve structural defects in the valve itself, *but result instead in right ventricular* failure and dilatation of the tricuspid ring

These lesions (1) may be left sided in origin, as in severe hypertension, coarctation of the aorta, or, rarely, aortic stenosis or mitral stenosis, (2) may occur at the level of the pulmonary vasculature, either in primary pulmonary vascular obstruction, or in pulmonary vascular obstruction associated with left to right shunts at the atrial, ventricular, or pulmonary artery levels, (3) may occur at the pulmonary valve level, as in severe pulmonic stenosis, with an intact ventricular septum, or (4) may affect the whole heart, as in prolonged arrhythmias, such as ectopic or paroxysmal atrial tachycardia, or in generalized myocardial disease, such as endocardial fibroelastosis

ACQUIRED LESIONS

As with the congenital lesions, the acquired ones may be divided into those causing structural deformity of the valve and those which simply cause dilatation of the tricuspid ring

LESIONS CAUSING STRUCTURAL DEFORMITY OF THE VALVE

1 Rheumatic heart disease with multivalvular involvement (by far the most important in this group)
2 Lupus erythematosis
3 Carcinoid tumors

LESIONS CAUSING DILATATION OF THE TRICUSPID RING

1 Any lesion which may cause left ventricular failure and secondarily right ventricular failure (for example severe hypertension from any cause coronary heart disease aortic valve disease—stenosis or regurgitation)
2 Cor pulmonale (either acute following pulmonary embolus or chronic associated with beryllіosis silicosis chronic bronchitis or emphysema)
3 Acquired pulmonic stenosi as in Bjork s syndrome (carcinoid with acquired pulmonic valve disease)
4 Lesions affecting the whole heart (such as rheumatic myocarditis bacterial endocarditis or other types of myocarditis)

AUSCULTATION (FIG 105)

FIRST HEART SOUND Since the principal valvular component in the first sound is due to mitral closure the intensity of the first sound varies according to the nature and severity of the underlying disease

PULMONIC SECOND HEART SOUND The pulmonic second sound is accentuated if tricuspid insufficiency is secondary to left sided failure or obstruction at the level of the pulmonary vasculature If there are obstructive

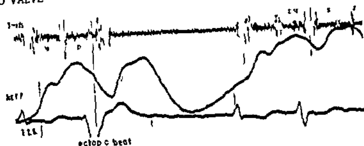


Fig 205 (L.L.S.B. Log 1) Plateau shaped c plus v wave in the jugular venous pulse secondary to ventricularization of the right atrial pressure curve in tricuspid insufficiency The first heart sound is variable in intensity and splitting and in its relation to the onset of the QRS complex of the electrocardiogram The first sound (1) in the first complex occurs 0 08 sec. after the onset of a QRS of 0 08 sec duration The sound is considerably accentuated because of a loud first valvular component It is split by 0 03 sec The first sound (1) in the second complex commences 0 14 sec after the onset of the QRS complex of the electrocardiogram This sound is again accentuated but this time it is the second valvular component which is accentuated The splitting is 0 04 sec The first sound in the third complex follows a QRS of normal duration The first sound in the third complex follows a long diastole and occurs 0 08 sec after the onset of the QRS The first sound in the fourth complex follows a short diastole and occurs 0 10 sec after the onset of the QRS complex

There is a holosystolic murmur (SM) of medium frequency and moderate intensity in each cycle The intensity of the murmur is greatly diminished with the ectopic beat The second sound (2) is variable in intensity It is moderately loud in the first cycle almost invisible in the second cycle and of normal intensity in the third and fourth cycles *It is interesting that in the second cycle when the second sound is almost invisible the preceding diastole equals that preceding the first complex where the second sound is normal in intensity* There is a diastolic murmur (DM) of low frequency and low intensity in each cycle The duration of systole and diastole varies from cycle to cycle The second sound is not clearly identifiable following the ectopic beat and systole cannot be measured but in the third cycle following a long pause systole is shorter (0 26 sec) than in the fourth cycle (0 30 sec) *The plateau* shaped c plus v wave of the jugular venous pulse occurs with the normally conducted beats and the ectopic beats Note the gradual descent of the v wave which does not begin to fall until after the second sound in any of the cycles except the last

lesions at the level of the pulmonic valve or isolated lesions of the tricuspid valve *itself* the pulmonic second sound is normal or diminished

MURMURS OF TRICUSPID INSUFFICIENCY (SEE P 272) No characteristic diagnostic murmurs accompany tricuspid insufficiency Usually, there is a blowing systolic murmur of moderate *intensity at the xiphoid process or* slightly to the left of the lower sternal border, sometimes it is harsh and *sometimes soft* This murmur was noted by Rosenback, in 1878, in dogs with injury to the tricuspid valve However, similar murmurs accompany many other cardiac lesions The murmur associated with tricuspid insufficiency unlike that of mitral insufficiency *is not transmitted to the left axilla* and left lung base but transmits well to the right lower part of the chest, *anteriorly It often* increases in intensity during inspiration

The murmur associated with tricuspid insufficiency is best *studied in* patients who have no other valvular involvement The phonocardiogram shows the murmur to be *decrescendo or plateau* shaped, it lasts throughout all of systole

A clinical point of some interest is the diagnosis of tricuspid insufficiency *in the presence of long standing, progressively severe mitral stenosis* Occasionally, in a patient who has been followed for years, an apical diastolic *murmur previously noted diminishes until it is barely audible, due to an increase in the severity of the stenosis, instead, a loud, or moderately loud, blowing systolic murmur may appear between the apex and the lower left sternal border* This suggests that as the mitral stenosis becomes severe, the right ventricle fails and tricuspid insufficiency results

In chronic rheumatic heart disease, there is no way of differentiating between true organic tricuspid insufficiency resulting from damage to the valve leaflets themselves, and functional tricuspid insufficiency, resulting from right ventricular failure and dilatation of the tricuspid ring By contrast, tricuspid insufficiency resulting from left ventricular failure or chronic cor pulmonale without rheumatic valvulitis, can be assumed to be functional

TRICUSPID STENOSIS

Tricuspid stenosis may be organic or functional and may be congenital or acquired

ORGANIC

CONGENITAL Congenital tricuspid stenosis almost always is associated with other major cardiac defects, and its diagnosis, except by finding a gradient across the tricuspid valve at cardiac catheterization, is virtually impossible

ACQUIRED Acquired organic tricuspid stenosis is usually due to rheumatic heart disease, in which case, the mitral valve certainly, and possibly also the aortic valve is involved Less commonly, the acquired lesions may be due to lupus erythematosis, bacterial endocarditis involving the tricuspid valve, or even tumors or thrombi in the right atrium In all these cases, there is true obstruction of the tricuspid valve orifice

Rheumatic tricuspid stenosis (Fig 106) Tricuspid stenosis should be suspected in patients who have rheumatic heart disease with mitral stenosis and aortic valve involvement, and who in the absence of any signs of acute rheumatic fever, have chronic congestive heart failure This is especially likely if the patient is a female, it is not likely however in patients who have rheumatic heart disease with severe mitral regurgitation

Rheumatic tricuspid stenosis is often accompanied by roentgenologic evidence of right atrial hypertrophy, and at least 25 per cent of the patients have atrial fibrillation

The clinical signs of rheumatic tricuspid stenosis are often dominated by those of concomitant mitral and aortic valve disease

Less common forms of tricuspid valve obstruction Ball valve thrombi (Fig 107) or tumors in the right atrium may cause mechanical obstruction

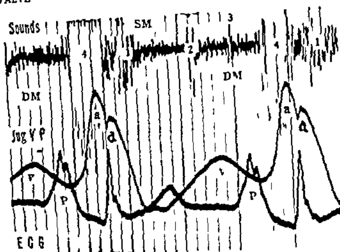


Fig 106 (L L S B Log) Very loud atrial sound in severe rheumatic tricuspid stenosis The tremendous presystolic activity is shown by the huge P waves of the electrocardiogram the tall a waves of the jugular venous pulse and the exceptionally loud atrial sound (4) on the phonocardiogram This sound commences 0 10 sec after the onset of the QRS complex It lasts at least 0 12 sec and greatly exceeds the other heart sounds in intensity The first sound (1) is of normal intensity and occurs 0 08 sec after the onset of the QRS complex There is a decrescendo holosystolic murmur (SM) of medium frequency and rather low intensity The second sound (2) is normal The diastolic murmur (DM) is of medium frequency and moderate intensity The third sound (3) is prominent and occurs 0 16 sec after the onset of the second sound

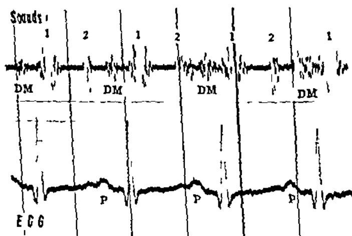


Fig 107 (L L S B Log) Phonocardiogram in unusual case of obstruction at the *tricuspid* valve secondary to a thrombus surrounding a piece of straw in the right *atrium* the mass being the site of infection for bacterial endocarditis This boy had a habit of chewing on a whisk broom and it is supposed that the straw passed *through the wall* of the esophagus and into the atrium Note the first (1) and second (2) sounds of normal intensity *and the somewhat* diamond-shaped diastolic murmur (DM) occupying mid and late diastole but with no presystolic crescendo The murmur is of medium frequency *and* low to moderate intensity As with many tricuspid diastolic murmurs an increase *in intensity with* inspiration can be noted from the beginning to the end of the tracing

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Organic

Congenital Congenital tricuspid stenosis almost always is associated with other major cardiac defects, and its diagnosis, except by finding a gradient across the tricuspid valve at cardiac catheterization, is virtually impossible

Acquired Acquired organic tricuspid stenosis is usually due to rheumatic heart disease, in which case, the mitral valve certainly, and possibly also the aortic valve is involved Less commonly, the acquired lesions may be due to lupus erythematosis, bacterial endocarditis involving the tricuspid valve, or even tumors or thrombi in the right atrium In all these cases, there is true obstruction of the tricuspid valve orifice

Rheumatic tricuspid stenosis (Fig 106) Tricuspid stenosis should be suspected in patients who have rheumatic heart disease with mitral stenosis and aortic valve involvement, and who, in the absence of any signs of acute rheumatic fever, have chronic congestive heart failure This is especially likely if the patient is a female, it is not likely however, in patients who have rheumatic heart disease with severe mitral regurgitation

Rheumatic tricuspid stenosis is often accompanied by roentgenologic evidence of right atrial hypertrophy, and at least 25 per cent of the patients have atrial fibrillation

The clinical signs of rheumatic tricuspid stenosis are often dominated by those of concomitant mitral and aortic valve disease

Less common forms of tricuspid valve obstruction Ball valve thrombi (Fig 107) or tumors in the right atrium may cause mechanical obstruction

an acquired lesion results from the rupture of an aneurysm of the sinus of Valsalva into the right atrium, but a more common cause is increased early diastolic filling of the right ventricle secondary to tricuspid insufficiency from any cause The congenital lesions are discussed in more detail in the section on congenital heart disease (see pp 306 311) but some aspects will be considered here With atrial defects and anomalous pulmonary venous drainage there may be a tremendous increase in diastolic blood flow through a normal or even through a dilated, tricuspid valve On auscultation there is a medium frequency, moderate intensity, mid and late diastolic rumbling murmur, but no presystolic crescendo (Fig 108)

JUGULAR VENOUS PULSE IN TRICUSPID VALVE DISEASE

When tricuspid valve disease is suspected careful examination of the jugular venous pulse may be rewarding (Fig 109) The examination should

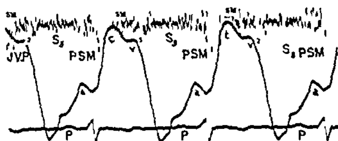


Fig 109 (L L S B Log) Plateau-shaped c plus v wave in tricuspid insufficiency in a 14 year old boy with rheumatic heart disease and a greatly enlarged heart secondary to free aortic regurgitation Note the prominent c plus v wave on the jugular venous pulse tracing with a rapid descent of the v wave early in diastole suggesting that there is no significant gradient across the tricuspid valve *Note* also the broad rather loud first sound (1) commencing 0 06 sec after the onset of the QRS complex the decrescendo systolic murmur (SM) of medium frequency and moderate intensity the normal second sound (2) the third sound (S3) of variable intensity and the middiastolic murmur with a prominent presystolic crescendo (PSM)

Systolic murmurs were present at both the apex and the xiphoid process There was no striking difference with respiration Because of the prominent jugular pulsation it was assumed that the systolic murmur at the lower left sternal border was due to tricuspid regurgitation

be performed with the patient seated or at least reclining at an angle of 45° from the horizontal Any visible pulsations of the neck veins in the seated patient should be regarded as abnormal, although in some short-chested, usually overweight persons, venous pulsations may be visible just above the right clavicle

Right ventricular failure with increased systemic venous pressure is the most common cause of jugular venous distention and the higher the systemic venous pressure, the higher in the neck can the pulsations be seen With greatly increased venous pressure and permanently distended veins,

at the tricuspid valve orifice If the tumor or thrombus is mobile, there will be an intermittent diastolic rumble at the xiphoid region, with changing qualities in the heart sounds, and perhaps attacks of syncope If the tumor or the thrombus is fixed in a position where it partially obstructs the tricuspid valve, the signs will not change and may resemble those of constrictive pericarditis When thrombi are present, rheumatic heart disease with atrial fibrillation usually is the underlying lesion In cases of tumor formation, the tricuspid valve obstruction may be present as an isolated lesion Rarely is the tricuspid valve the site of bacterial endocarditis (Fig 107)

AUSCULTATION At the xiphoid region there is a mid diastolic, and possibly a presystolic, rumbling murmur This can rarely be clearly distinguished from a simultaneous apical rumble of mitral stenosis, but it may be suspected because of its accentuation with inspiration

Rivero Carvallo[3] noted that the murmur of tricuspid stenosis becomes considerably stronger following deep inspiration This phenomenon can be explained by the increase in the size of the chest cavity following inspiration, causing an increase in intrathoracic negative pressure and resulting in greatly increased venous inflow through the right side of the heart

Recently, Kossman[4] described the presence of the opening snap of the tricuspid valve in tricuspid stenosis

FUNCTIONAL

Functional tricuspid stenosis (Fig 108) is that which is due to increased blood flow through a normal or even a dilated tricuspid orifice It may be congenital, resulting from left to right shunts through atrial defects or via total or partial anomalous pulmonary venous drainage, or acquired Rarely,

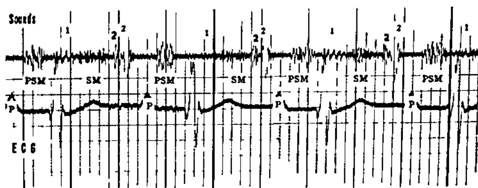


Fig 108 (L L S B Log) A late diastolic presystolic murmur frequently heard in patients having total anomalous pulmonary venous drainage and presumably due to greatly increased flow through the tricuspid valve This represents a relative degree of tricuspid stenosis where the valve itself is anatomically normal Note the very low intensity first sound (1) the holo systolic murmur (SM) of low intensity and medium frequency the well split (0 04 sec) second sound (2 ") with the second component at the split much louder than the first and the pre systolic murmur (PSM) not crescendic up to the first sound but somewhat diamond shaped commencing 0 08 sec after the onset of the P wave and continuing for 0 12 sec to the onset of the QRS complex

looked This sign, although easy to elicit in children and in thin adults, may be difficult to detect in obese persons Vigorous pulsations of the abdominal aorta may cause transmitted pulsations and further confuse the examiner

PRESYSTOLIC HEPATIC PULSATIONS

Tricuspid valve obstruction or increased right ventricular end diastolic pressure necessitates increased atrial activity to fill the right ventricle Because of the absence of valves in the great veins entering the right atrium a reflux of blood may cause a pressure wave to be transmitted to the liver Such a wave is presystolic in timing and coincides with the *a* wave of the jugular venous pulse Many patients with advanced tricuspid stenosis have atrial fibrillation, in which case, presystolic hepatic pulsations are not present

Adults with tricuspid insufficiency and tricuspid stenosis, or right ventricular failure could have both presystolic and systolic pulsations

DIFFERENTIAL DIAGNOSIS It is important to realize that a presystolic liver pulsation is not necessarily diagnostic of tricuspid valve obstruction, and that it may occur with any type of right ventricular failure or obstruction which interferes with the filling of the right side of the heart We have observed it in cases of subendocardial sclerosis, pulmonary vascular obstruction, pure pulmonic stenosis constrictive pericarditis, and pericardial effusion

SYSTOLIC HEPATIC PULSATIONS

Although systolic pulsations of the liver or the deep veins in the neck may be indicative of tricuspid insufficiency nevertheless, tricuspid dilatation may be secondary to generalized cardiac failure, and not related specifically to primary disease of the tricuspid valve

The systolic hepatic pulsation in tricuspid regurgitation corresponds to the *c* plus *v* wave plateau in the right atrial pressure curve

CONCLUSIONS

It would seem that the diagnosis of tricuspid valve disease is frequently overshadowed by concomitant mitral and aortic lesions in those cases which are rheumatic in origin When tricuspid lesions are secondary to nonvalvular heart disease such as chronic cor pulmonale, the detection is somewhat easier Careful examination of the jugular venous pulse and the liver pulsations may be rewarding

REFERENCES

1 Smith J A and Levine S A The clinical features of tricuspid stenosis A study of trivalvular stenosis Am Heart J 23 739 1942

there may be little or no pulsation, and in *extremely obese persons it may* be necessary to examine the veins under the tongue to appreciate fully the degree of distention

If free tricuspid regurgitation exists, the veins in the neck may be completely flat during diastole and fill with a sustained reflux during systole

With severe tricuspid stenosis and normal sinus rhythm, or in tricuspid atresia, the jugular venous pulsation is presystolic and reflects increased right atrial activity If atrial fibrillation is present, no presystolic pulsation occurs

A late diastolic filling wave can be seen in congestive failure with a slow heart rate Because of the prolonged diastole, the right ventricle, then the right atrium, and finally the neck veins fill, giving a positive wave late in *diastole*

The positive systolic wave in tricuspid insufficiency coincides with the *c* plus the *v* wave of the normal jugular venous pulse It is said that in tricuspid stenosis combined with tricuspid insufficiency, the rise in the systolic plateau is more gradual and the fall occurs later than in pure tricuspid insufficiency,[5] but we have not found this statement to be infallible

In cases of severe tricuspid stenosis there may be a gradual decline from *the summit of the v wave of the jugular venous pulse following the opening* of the tricuspid valve, and Gibson and Wood [6] stated that, even in the presence of atrial fibrillation, this descent of the *v* wave is too gentle to admit any other diagnosis

In pure tricuspid insufficiency there is a rapid descent of the *v* wave immediately following the opening of the tricuspid valve, since there is no obstruction to blood flow through the tricuspid orifice, and the pressure in the right atrium and in the veins of the neck falls rapidly

It is important to note that a large *a* wave may be present in any patient who has right ventricular failure and an elevated end diastolic pressure in the right ventricle This wave is a reflection of the right atrial activity needed to exceed this elevated pressure

Differential diagnosis

In studying congested neck veins, it is necessary to consider constrictive pericarditis, pericardial effusion, and the superior mediastinal syndrome, in addition to congestive heart failure and tricuspid valve disease in the differential diagnosis

LIVER PULSATIONS IN TRICUSPID VALVE DISEASE

Although enlargement of the liver is a well known sign in congestive heart failure, and liver size is *carefully measured in all patients with this condition*, nevertheless, true expansile pulsations of the liver, which may be helpful diagnostically, are much less carefully sought and are frequently over

section IV

rheumatic heart disease

2 Rosenback, O Der Mechanismus und die Diagnose der Mageninsufficienz Samml klin Vortr (Innere Med No 51), Leipzig 1878, p 1315
3 Rivero Carvallo J M Signo para el diagnostico de la tricuspidea Arch Inst cardiol Mexico 16 531 1946
4 Kossman C E The opening snap of the tricuspid valve, Circulation 11 378, 1955
5 Messer, A L Hurst, J W, Rappaport M B and Sprague, H B A study of the venous pulse in tricuspid valve disease Circulation 1 388, 1950
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16 / Acute Rheumatic Carditis

In these days of long term antibiotic prophylaxis for the prevention of recurrent streptococcal infections and of the treatment of acute infectious states to avoid bacterial endocarditis, it is imperative that we be able to decide whether the murmurs in acute rheumatic fever are due to true valvular disease or are of the so-called "innocent," "functional," or "benign" types

Since there is no specific diagnostic test for rheumatic fever, and since the important fact from a prognostic point of view is to know whether or not the patient has myocardial and endocardial involvement auscultation of the heart is of the utmost importance Confirmatory evidence may be the presence of a pericardial friction rub, definite cardiac enlargement, the onset of congestive heart failure or the presence of suggestive electrocardiographic changes

In acute rheumatic fever with carditis, the lesions may involve the aortic valve, the mitral valve, or both There may or may not be associated cardiomegaly In severe cases of rheumatic pancarditis with congestive heart failure there may be evidence of tricuspid insufficiency

It is important to realize that in acute rheumatic fever the mitral valve is the valve most commonly affected, and the aortic valve less commonly the valves on the right side of the heart are much less frequently affected than those on the left In a study of 100 patients who died from rheumatic disease before the age of twenty-one years Bland[1] cited the valve involvement as follows Mitral, 98 aortic, 71 tricuspid 31 pulmonary 5, no valvular involvement, 1

AUSCULTATION

First heart sound

First degree block If the P R interval is prolonged, the first heart sound may be diminished in intensity One reason may be that in the normal first heart sound the atrioventricular valves are pushed well down into the ventricle by the atrial contraction, and they have a considerable distance to travel when tensing and closing With a prolonged P R interval, the valves tend to float up to a more closed position because of the eddy currents which form under the valves as a result there is less of a snap with the tensing of the valve

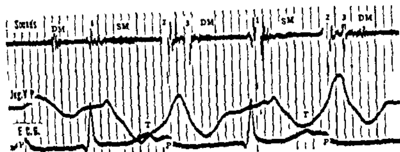


Fig 111 (Apex Log) Effects of a prolonged P R interval on the heart sounds in acute rheumatic fever The beginning of each first sound complex commences 0 04 sec after the onset of the QRS The sound in the first complex consists of two low intensity peaks split by 0 04 sec with the first group being the more prominent In the second cycle the first sound (1) again consists of two peaks but this time the second peak is the apex of a group of extremely high intensity high frequency vibrations The low intensity systolic murmur (SM) is little changed and occupies the first two thirds of systole Note the moderately loud second sound (2) in both cycles In the first cycle the third sound (3) is slightly louder than it is in the second cycle although the position of the P waves in relation to this sound is the same The diastolic murmur (DM) follows the P wave wherever it falls in the cycle and so precedes the first sound in the first cycle and falls in its expected position following the third sound in the two succeeding cycles

blowing in character and frequently soft It is well conducted to the left axilla and the left lung base, which is expected from the anteroposterior direction of the blood flow from the left ventricle to the left atrium The murmur may lessen in intensity with deep inspiration, but it does not disappear If the patient changes from a recumbent to a sitting posture the murmur remains fairly constant

Tricuspid systolic murmur (see pp 221 224)

Although, occasionally the tricuspid valve is involved during the inflammatory process of acute rheumatic pancarditis, tricuspid systolic murmurs usually are the result of tricuspid insufficiency secondary to generalized

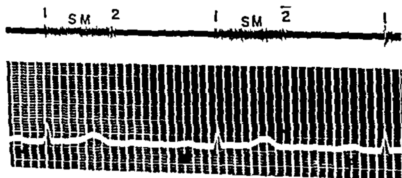


Fig 112 (Apex Log) Mitral insufficiency in acute rheumatic heart disease Note the P R interval of 0 18 sec the low intensity first sound (1) 0 02 sec after the onset of the QRS complex the holosystolic murmur (SM) of medium frequency and low to moderate intensity and the low intensity second sound (2)

An alternative explanation is that normal atrial systole almost completely closes the valve by the "closing jet" mechanism In some way this more efficient manner of closure is interfered with when atrial systole occurs considerably in advance of ventricular systole, permitting a blurring of the first sound either because of some insufficiency when the ventricle contracts, or because the leaflets have decreased mobility in this abnormal atrioventricular relationship

A prolongation of the P-R interval may be suspected if the first heart sound is diminished or if the *a* waves are clearly distinct from the *c* waves in the jugular venous pulse tracing

If the P R interval exceeds 0 30 sec , the first heart sound may again return to normal intensity

SECOND DEGREE BLOCK (FIGS 110 AND 111) Heart sounds vary, depending on the lengthening of the P-R interval In Wenckebach's phenomenon, the successive first heart sounds may diminish progressively in intensity until a beat is dropped When the P R interval increases sufficiently, a summation gallop may result In 2 1, 3 1, or 4 1 block, the first, second, and third heart sounds may all vary, depending on the relationship of the atrial systole to each of these sounds

APICAL SYSTOLIC MURMUR

The murmur most commonly encountered in acute rheumatic fever is the apical systolic murmur of mitral insufficiency (Figs 112 and 113) This murmur has certain definite characteristics It is heard best at the point of maximal impulse and is grade 2 or more in intensity Rarely is it louder than grade 4 in the acute stage The murmur commences with the first sound, which it often obscures, and continues throughout most of systole It is

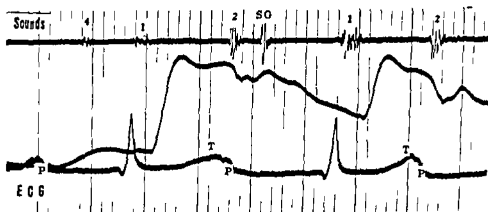


Fig 110 (Apex Log) Varying relationship of the P waves to the QRS complex in the electrocardiogram causing changes in the intensity of the heart sounds Note the low intensity first heart sound (1) in the first cycle and the much more intense first sound (1) in the second cycle The second sound (2) is of similar intensity in each cycle The third sound combines with the atrial sound to form a summation gallop (SG) in the first cycle but not in the second There is a fourth sound (4) following the first P wave

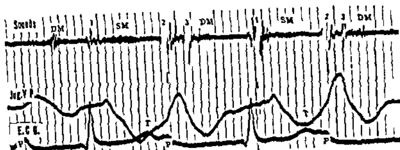


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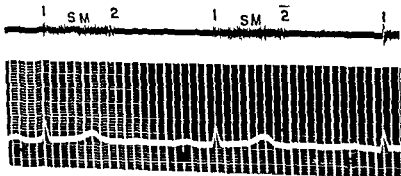


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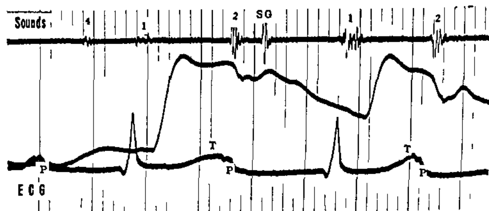


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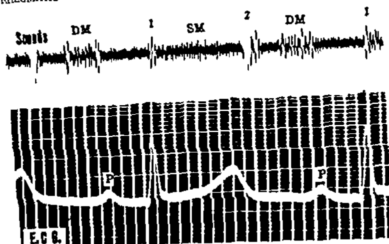


Fig 114 (Apex Log) Apical systolic and apical diastolic murmurs three months after an attack of chorea without apparent cardiac involvement Note the normal first sound (1) 0 04 sec after the onset of the Q wave the systolic murmur (SM) of medium frequency and moderate intensity the narrowly split second sound (2) of normal intensity and the mid diastolic murmur (DM) of medium frequency and moderate intensity

actual inflammatory thickening of the mitral leaflets and thus some resistance to the left ventricular inflow may result

This murmur is often heard during the acute phase of rheumatic fever, and, in our experience, it is always accompanied by the apical systolic murmur of mitral insufficiency It does not occur as an isolated phenomenon The probable reason for this is that the mitral insufficiency causes increased left atrial pressure and increased left atrial blood volume, thus, the early rapid filling of the left ventricle occurs with a greater than normal pressure gradient and a greater than normal blood flow, thereby accentuating the relative mitral stenosis

When apical systolic and diastolic murmurs are present together, the term 'mitral valve involvement' is frequently used The apical diastolic murmur noted under these conditions may disappear completely later on or it may persist with the development of true organic mitral stenosis Occasionally the murmur may remain even without true organic mitral stenosis if the degree of mitral regurgitation is severe The diastolic murmur commences just before the third heart sound and continues into mid diastole Under these circumstances the third heart sound may be loud and the rapid inflow wave of the apex linear cardiogram may be pronounced Sometimes the diastolic murmur may be of short duration and may be masked by the loud third heart sound

AORTIC DIASTOLIC MURMUR (SEE PP 200 206)

The high pitched, soft, blowing diastolic murmur of aortic insufficiency, best heard in the aortic area or along the left sternal border in the third and

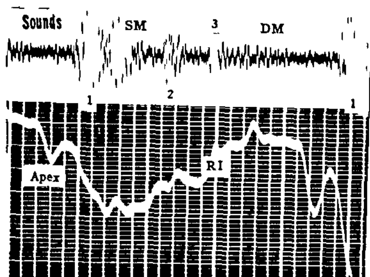


Fig 113 (Apex Log and linear) Apical systolic and apical diastolic murmurs in a case of fatal rheumatic pancarditis Note the *relatively normal first (1) and second (2) heart sounds* and a prominent third sound (3) the decrescendo systolic murmur (SM) of moderate intensity and medium to high frequency and the long diastolic murmur (DM) of low intensity and medium frequency There is no opening snap and no presystolic crescendo The apical diastolic murmur in this case is not indicative of organic mitral stenosis but merely represents the type of diastolic murmur heard with relative mitral stensosis secondary to left ventricular dilatation or associated with mitral regurgitation

rheumatic myocarditis, and are associated with congestive heart failure The accompanying murmur is heard best either to the right or the left of the sternum in the fourth or fifth intercostal spaces Sometimes it is heard best between the apex and the lower left sternal border, where it may be confused with the murmur of mitral insufficiency which usually is present *at the same time*

A helpful sign in further establishing the diagnosis of tricuspid insufficiency may be the presence of a sustained deep systolic venous pulsation in the neck It is important to realize that tricuspid valvular disease does not occur as an isolated phenomenon in rheumatic heart disease Discovery that *the tricuspid valve is involved in the rheumatic process automatically implies the additional involvement of the valves on the left side of the heart*

APICAL DIASTOLIC MURMUR (FIG 114)

Along with the mitral regurgitant murmur of acute rheumatic fever there may be an apical mid diastolic rumble This murmur is not preceded by an opening snap and is not terminated by a presystolic crescendo Obviously it is not caused by true organic mitral stenosis, *it may be caused by several* factors, of which two appear most probable First, because of the left ventricular dilatation that may accompany the acute myocarditis of rheumatic fever, it is possible that, even with a normal mitral valve, the blood flow into a dilated chamber could result in relative mitral stenosis, and so cause an *apical diastolic* rumble Second, the acute endocarditis may cause an

fever It may become so severe that the development of a giant left atrium follows, or it may be so mild that it causes the patient no difficulty at all The intensity of the murmur, although helpful, by no means indicates the exact degree of mitral insufficiency

Mitral stenosis does not develop as an acute lesion At least three to five years, and often as many as thirty to forty, may elapse before significant stenosis develops

REFERENCE

1 Bland E F and Jones T D Fatal rheumatic fever Arch Int Med 61 161 1938

fourth interspaces, is common in acute rheumatic fever *Frequently it is missed because of the lack of care in auscultation* This murmur may be only grade 1 or grade 2 in intensity It is soft, is easily masked by the breath sounds, and is often of extremely short duration, lasting no longer than one fourth to one third of diastole It starts with the second heart sound and is usually *decrescendo* in configuration, but occasionally it may have an early short crescendo phase, followed by a longer decrescendo phase The essential point in detecting this murmur is careful listening with the diaphragm chest piece while being sure that the patient holds his breath during auscultation

In adults this murmur is best heard with the patient leaning forward and to the left, holding the breath in full expiration Occasionally, it may be *necessary for the patient to assume the knee chest or the knee elbow position before the murmur can be detected*

In children, the murmur often is heard best with the child lying supine Frequently, it is heard better along the left sternal border than in the aortic area

A diaphragm chest piece must be used to screen out the low frequency vibrations, thus isolating the more brilliant high frequency components If *a bell chest piece is used alone, this murmur will be missed frequently*

Natural history of murmurs of the mitral valve with rheumatic involvement (see pp 171 197)

Rheumatic heart disease involving the mitral valve is a dynamic process Over periods as long as forty years or even longer, the mitral valve may show successively (1) a mitral systolic murmur, suggesting mitral insufficiency, predominantly, (2) mitral systolic and diastolic murmurs, suggesting mitral insufficiency, possibly with relative mitral stenosis and later with true organic mitral stenosis, and (3) a mitral diastolic murmur, suggesting mitral stenosis, predominantly

In the early stages of the disease, the mitral murmurs are either purely *systolic or systolic and mid diastolic, but they are never purely diastolic* The diastolic murmur of predominant rheumatic mitral stenosis is rarely seen in patients younger than nine or ten years of age We have seen tight mitral stenosis with diastolic murmurs only in some patients from ten to thirteen years old, but such murmurs are more common among patients fifteen to *thirty five years of age or older*

Combined mitral stenosis and mitral regurgitation may be noted any time after the age of approximately five years, that is to say, two to three years after the earliest expected onset of rheumatic fever On the other hand, mitral systolic and diastolic murmurs reflecting only "mitral valve involvement" *may be noted any time after the onset of acute rheumatic carditis*

Pure mitral regurgitation may be the only lesion following rheumatic

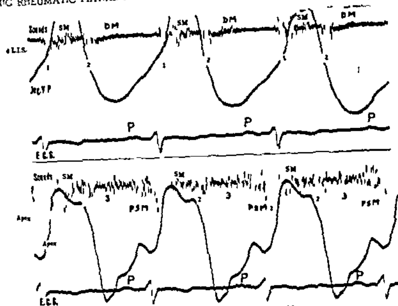


Fig 115 (Upper trac ng 4 L I S Log Lower tracing Apex Log) Decrescendo aort c d astol c murmur in the upper trac ng reco ded at the fo th left interspace M d d astol c murmur w th a presystol c crescendo in the lower tracing recorded at the apex showing the d fference in conf gurat on of these two mu mu s Note the f rst heart sound (1) of normal *inte s ty 0 09 sec after the onset of the* QRS complex the decrescendo systol c murmur (SM) in each trac g loude at th ap x than at the aort c area the normal seco d hea t sound (2) the decrescendo d astol c murmur (DM) regula in configurat on and of med um h gh frequency beg nn ng with *th second he rt sound in the uppe trac ng and the m d* d astol c murmur w th a p syst l c cr scendo (PSM) in the lower trac ng This mu mur comme ces as a series of low f equency v b at ons at the t me of the th rd sound (3) and s c esc d c up to the succeed g first heart so d



Fig 116 (Apex L g nd l a) Ap cal sy tol c and ap cal d ast l c mu murs in a com b ned les on of mit al stenos s and regurgitation together with atrial f b illat on Note the extremely lo d f st sound (1) 0 11 sec fter the onset of the QRS complex the holosystol c murmur (SM) of h gh intens ty and med um fr q ency the lo d seco d he rt sound (2) th loud op ni g sn p of the m t al lve (OS) co c d ng w th the valley o O po nt of the apex l nea card ogram (m ddle trac ng ma k d Apex) the loud thi d h *art s und (3) co* inc d ng w th the apex of the rap d nflow w e (RI) of the apex l near ca d ogram a d the somewhat cresce do d crescendo diast l c m m (DM) of low to med um frequency and moderate i tens ty tr l ng off bef re *the succeed g f rst sound*

17 / Chronic Rheumatic Mitral Insufficiency

AUSCULTATION

The sounds and murmurs of chronic rheumatic heart disease (Figs 115, 116, and 117) are principally those caused by lesions of the mitral and aortic valves (see pp 234 235, 237, and 238), and, to a lesser extent, the tricuspid and pulmonic valves (see pp 233 and 235 236)

The lesions of the aortic and mitral valves *may be those of insufficiency*, of stenosis, or both Of the tricuspid valve, the lesion is usually insufficiency, and more rarely stenosis, of the pulmonic valve it is almost always insufficiency Various combinations of these murmurs may be present

First heart sound

While it is true that the first heart sound may be diminished in mitral insufficiency, especially in the presence of first degree heart block, more commonly, the vibrations of the first heart sound appear normal on the phonocardiogram, but the sound may be masked by the systolic murmur In chronic cases, associated with some increased rigidity of the mitral leaflets, the first heart sound is often accentuated

Second heart sound

Mitral insufficiency may result in increased left atrial pressure, increased pulmonary capillary pressure, and, consequently, in pulmonary artery hypertension As a result, the second heart sound may be slightly or even markedly accentuated

Murmur of mitral insufficiency

As previously described (pp 234 235), the murmur of mitral insufficiency (Fig 118) is heard best at the apical region, it is blowing in character, is continuous throughout systole, often masks the first sound, transmits well to the left axilla and left lung base, is grade 2 or more in intensity, and does not disappear with respiration or changes in position The murmur may represent an isolated lesion, especially in young women, who tend to develop a true giant left atrium The murmur may be soft in character, it may be harsh and rough, associated with a thrill, or it may be high pitched, almost musical or whistling, a sound which some people have compared with various bird calls, such as that of the seagull

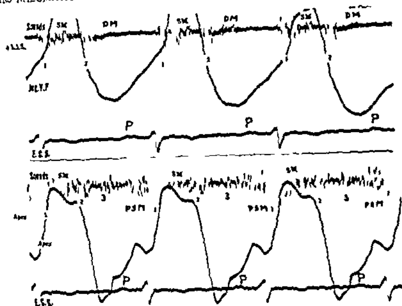


Fig 115 (Upper tracing 4 L.I.S. Log Lower tracing Apex Log) Decrescendo aortic diastolic murmur in the upper tracing recorded at the fourth left interspace Mid diastolic murmur with a presystolic crescendo in the lower tracing recorded at the apex showing the difference in configuration of these two murmurs Note the first heart sound (1) of normal intensity 0.09 sec after the onset of the QRS complex the decrescendo systolic murmur (SM) in each tracing louder at the apex than at the aortic area the normal second heart sound (2) the decrescendo diastolic murmur (DM) regular in configuration and of medium high frequency beginning with the second heart sound in the upper tracing and the mid diastolic murmur with a presystolic crescendo (PSM) in the lower tracing This murmur commences as a series of low frequency vibrations at the time of the third sound (3) and is crescendic up to the succeeding first heart sound

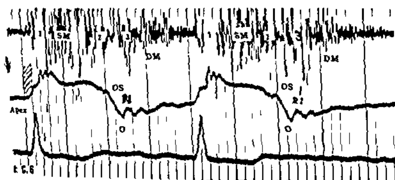


Fig 116 (Apex Log and linear) Apical systolic and apical diastolic murmurs in a combined lesion of mitral stenosis and regurgitation together with atrial fibrillation Note the extremely loud first sound (1) 0.11 sec after the onset of the QRS complex the holosystolic murmur (SM) of high intensity and medium frequency the loud second heart sound (2) the loud opening snap of the mitral valve (OS) coinciding with the valley O point of the apex linear cardiogram (middle tracing marked Apex) the loud third heart sound (3) coinciding with the apex of the rapid flow wave (RI) of the apex linear cardiogram and the somewhat crescendo decrescendo diastolic murmur (DM) of low to medium frequency and moderate intensity trailing off before the succeeding first sound

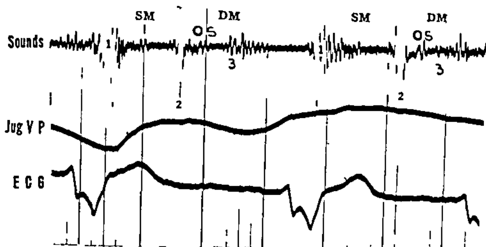


Fig 117 (Apex Log) Atrial fibrillation with marked intraventricular conduction delay in rheumatic heart disease with mitral stenosis Note the QRS complex with a duration of 0 15 sec the normal intensity first sound (1) not split and with the maximal vibrations 0 13 sec after the onset of the Q wave the narrow slightly accentuated second sound (2) the low intensity opening snap (OS) 0 08 sec after the second sound the third sound (3) of low intensity and low frequency and the diastolic rumble (DM) of low frequency and low intensity

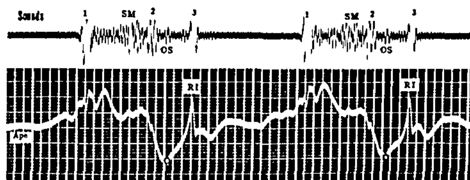


Fig 118 (Apex Log and linear) Mitral insufficiency with a loud apical systolic murmur and mild mitral stenosis as shown by the opening snap of the mitral valve and the loud third heart sound Note the first sound (1) of normal intensity the holosystolic murmur (SM) of medium frequency and moderate intensity the second sound (2) of normal intensity the opening snap (OS) corresponding with the valley or O point in the apex cardiogram (that is the beginning of the rapid inflow wave) and the loud third sound (3) preceded and followed by a few very low intensity vibrations and occurring at the apex of the rapid inflow wave (RI)

It is well to state here that all apical systolic murmurs are not due to mitral insufficiency, and conversely, severe mitral insufficiency has been found at surgical exploration when there was no apical systolic murmur Apical diastolic murmurs may be heard with mitral insufficiency Left atrial distention resulting from the mitral reflux, together with the normal left atrial inflow from the pulmonary veins, results in an increased diastolic flow across the mitral valve, and a diastolic murmur of relative mitral stenosis may be noted

IMPORTANCE OF DIAGNOSING MITRAL INSUFFICIENCY

At one time mitral insufficiency was by far the most commonly diagnosed of all cardiac lesions The diagnosis was based on the presence of an apical systolic murmur Examination of large numbers of soldiers during World War I drew attention to the good health of the majority of persons with lesser grades of apical systolic murmurs, and slowly the pendulum swung the other way until mitral insufficiency was regarded by some as being an extremely rare condition As usual, the truth seems to lie between the two extremes, and mitral insufficiency is a fairly common entity in any rheumatic fever hospital, both in the wards for patients with acute rheumatic fever and in the outpatient departments

With the increasing interest in the surgical correction of severe mitral insufficiency, it is becoming more and more important to be able to assess with some accuracy the degree of insufficiency

In pure mitral insufficiency, the left atrial enlargement seen on the roentgenogram and the apical systolic murmur heard at auscultation are complementary methods of diagnosis In the presence of mitral stenosis, however, the roentgenologic sign of left atrial enlargement is of no value in assessing the degree of mitral insufficiency as the enlargement could well be due to the mitral stenosis alone Hence we are left to evaluate the murmur on its own merits

The absence of a 'significant' (grade 2 or greater) apical systolic murmur does not exclude the presence of slight or even severe, mitral insufficiency Most clinicians are familiar with cases in which a diagnosis of mitral stenosis was made because the only murmur present was an apical diastolic murmur, and in which, after thoracotomy had been performed to correct this suspected mitral stenosis careful search of the precordium did not reveal an apical systolic murmur, even though a marked regurgitant jet had been felt at surgery

In general, the apical systolic murmurs associated with mitral insufficiency vary in intensity and duration, depending on the degree of insufficiency On the phonocardiogram, the murmur may be plateau shaped, continuing from the first heart sound to the second heart sound, but more commonly it is decrescendo following the first heart sound Occasionally it is crescendo beginning in early systole and continuing to the second heart sound

It is fairly safe to assume that an apical systolic blowing murmur which is grade 3 or louder and is well conducted to the left axilla and the left lung base is due to mitral insufficiency, murmurs of grade 2 with similar characteristics probably are due to mitral insufficiency, and murmurs less than grade 2 may be secondary to mitral insufficiency, but also may be caused by a variety of other factors both intracardiac and extracardiac Slight degrees of mitral insufficiency may be missed by using these criteria too rigidly, however, if less strict criteria are used, mitral insufficiency may

often be diagnosed where it does not exist Apical systolic murmurs occupying the latter parts of systole are common in normal persons, and grade 1 or grade 2 murmurs are frequently heard in persons with no apparent heart disease Since minor degrees of mitral insufficiency are tolerated without obvious difficulty, it would appear preferable to underdiagnose rather than overdiagnose this condition

The prognosis for persons who have a grade 2 or greater apical systolic murmur during the acute attack of rheumatic fever has been demonstrated by Bland and his associates[1] in a twenty year follow up study of 87 such patients In 29 (33 per cent) potential rheumatic heart disease was indicated, 35 (40 per cent) had mitral insufficiency, 16 (19 per cent) had mitral insufficiency combined with mitral stenosis, and 7 (8 per cent) had mitral stenosis alone Of these 87 patients, 9 (10 per cent) had died

Diagnosis of mitral insufficiency in the presence of aortic stenosis

In adults, it may be difficult to diagnose mitral insufficiency in the presence of aortic stenosis because the systolic murmur of aortic stenosis may also be heard well at the cardiac apex The areas of transmission of an apical systolic murmur may be very helpful in deciding whether the murmur is mitral or aortic in origin If an apical systolic murmur is well heard in the left axilla and left back and not in the aortic area, it is almost always mitral in origin, but if it is well heard at both the aortic area and the apex and not in the left lung base, it is much more likely to be due to aortic stenosis In many cases, the lesions are combined In children, however, the situation is not so difficult, as the aortic valve murmur seems to be more "stenotic" in character, also, congenital aortic stenosis usually is an isolated lesion

In cases of marked aortic insufficiency, true mitral insufficiency is likely to be present Often there is cardiomegaly secondary to left ventricular dilatation, with resultant stretching of the mitral ring To make the diagnosis even more confusing under these conditions, either an apical mid diastolic or a true Austin Flint presystolic murmur may be present, suggesting mitral stenosis in addition to the mitral insufficiency

REFERENCE

1 Bland E F and Jones T D Rheumatic fever and rheumatic heart disease A twenty year report on 1000 patients followed since childhood Circulation 4 836 1951

2 Bland E F Jones T D White P D Disappearance of physical signs of rheumatic heart disease *J A M A 107 569 73 1936*

section V

congenital heart disease

INTRODUCTION

A careful study of the heart sounds and murmurs in congenital heart disease may be of considerable assistance in diagnosis Many congenital heart defects are accompanied by murmurs so characteristic that the diag nosis is immediately obvious, others are sufficiently suggestive to give good clinical directives There are certain general rules worth mentioning, but, like all generalities, they must not be followed too rigidly Some are as follows

1 Harsh basal murmurs which are present at birth and which persist almost invariably originate from semilunar valve stenosis either pulmonic or aortic

2 Mid diastolic murmurs at the apex or the lower left sternal border usually are asso ciated with a sizable left to right shunt less commonly they indicate mitral valve disease

3 A booming second sound at the pulmonary area is consistent with hypertension of the pulmonary artery and a second sound of diminished intensity in this region suggests the presence of pulmonary stenosis

4 *Continuous murmurs which are heard in the first 24 to 48 hours of life* and then dis appear may be due to a ductus arteriosus remaining patent for one or two days after birth

5 Continuous murmurs heard in the first few weeks of life usually are not due to patent ductus arteriosus

6 Small ventricular septal defects and mild infundibular pulmonic stenosis frequently cannot be distinguished from one another by auscultation

7 Systolic murmurs up to grade 2 in intensity occurring in the lower left sternal border are common but frequently they are not associated with any significant heart disease

8 Blue babies with well split second sounds do not have tetralogy of Fallot

9 Blue babies with mid diastolic rumbles probably do not have transposition of the great vessels

Under the various abnormalities to be described, the reasons for these generalities will be discussed in some detail

18 / Patent Ductus Arteriosus with a Differential Diagnosis of Continuous Murmurs

The typical murmur of patent ductus arteriosus is continuous, with a machinery quality, it must be differentiated from to-and fro murmurs and from other continuous murmurs which do not have a machinery quality

Although Gibson [1] described the murmur in 1900, the diagnosis of patent ductus arteriosus was of little more than academic interest until Gross developed a successful operation for its correction in 1939 [2] Subsequently, wide experience has shown that there is tremendous variation in the accompanying murmurs and sounds

Since, under ordinary circumstances, the pressure in the aorta exceeds that in the pulmonary artery throughout the cardiac cycle (Fig 119), blood flow across a patent ductus arteriosus is continuous throughout systole and diastole If sufficient turbulence results when this flow encounters the usual forward flow through the pulmonary valve, a continuous murmur is audible (Fig 120)

In infants with rapid heart rates, the shortened diastole makes it more

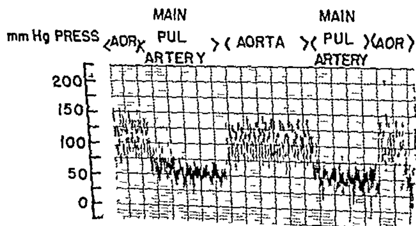


Fig 119 P ess trac gs result ng fr m the passage of a card ac catheter from aorta to pulm n ry artery to aort to p lmonary artery to aorta Both the systol c and d astol c pres su es in the aorta a e h gher than th co respond ng pressu es in the pulmonary artery As a esult flow from the aort to the p lmona y artery can occur throughout the card ac cycle g ing ise to a continu us murm r (Pressu e tr c ng court sy of Dr Ab aham R dolph Ca d op lmonary Labo atory Ch ldren s Med cal Cent r Boston M ss)

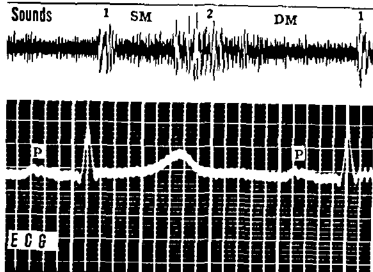


Fig 120 (2 L I S Log) Typical continuous machinery murmur of patent ductus arteriosus The normal first heart sound (1) occurs 0 07 sec after the onset of the QRS complex the second heart sound (2) is not clearly distinguishable and there is a continuous systolic diastolic murmur (SM DM) with the maximal intensity occurring about the expected time of the second sound The murmur is of medium frequency and moderate intensity

difficult to hear diastolic murmurs, and the typical machinery effect may not be present even though both systolic and diastolic components are audible If there is only a small gradient during diastole (Fig 121), there may be too little flow to cause a murmur, and if there is a large ductus, with a

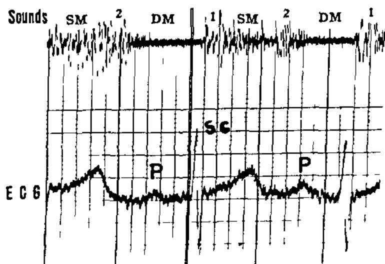


Fig 121 (2 L I S Log) Systolic murmur with short early diastolic murmur in a patient age 3 months with patent ductus arteriosus Note the apparently split first heart sound (1) with a normal intensity the first component occurs 0 05 sec after the onset of the QRS complex and the very loud second component (SC) 0 09 sec after the onset of the QRS complex almost certainly represents a systolic ejection sound Note also the holosystolic murmur (SM) of medium frequency and moderate intensity the second sound (2) of variable intensity and the short early decrescendo diastolic murmur (DM) of medium to high frequency

diameter comparable to that of the aorta and the pulmonary artery, sufficient turbulence for the development of a murmur may not occur

AUSCULTATION

First heart sound

In patent ductus arteriosus the first heart sound is of normal intensity and is not delayed It is heard best at the apex or the lower left sternal border and may not be audible at the pulmonic area, where it is often masked by the loud continuous murmur In patients who have congestive failure secondary to the large left to right shunt, it may be diminished

Second heart sound

The second sound at the pulmonary area is often masked by the continuous murmur, however, auscultation at the left sternal border, just below the site of maximal intensity of the murmur, may reveal a clearly audible or even an accentuated second sound (Hochsinger's sign), with more or less splitting than normal (Fig 122)

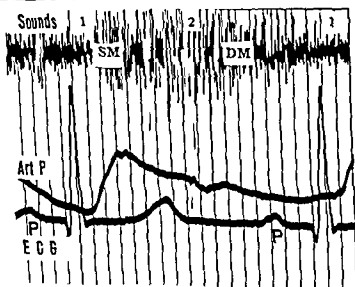


Fig 122 (2 L I S Log) Lo d rough cont ous systol c and d astol c mu murs show g a p om nent seco d h rt sou d Note the fi st heart sound (1) of low to moderate intensity occu ring 0 08 sec after the o set of th QRS compl the ext emely loud seco d sound (2) nd the h gh t ns ty regula systolic d astol c cont n ous mu mu (SM DM) with m mum ntens ty t wa d the latte pa t of systole and the early part of diastole

Intensity The intensity of the second sound depends on the pressure in the pulmonary artery and this of course is dependent on both pulmonary blood flow and pulmonary vascular resistance There are two obvious causes for increased prominence of the pulmonic second sound (1) increased

pressure in the pulmonary artery and (2) the obliteration of the continuous murmur, with its masking effect on the second sound, if pulmonary vascular obstruction increases and the left to right shunt decreases

SPLITTING Because of the increased left ventricular output with large shunts, left ventricular systole may be prolonged, and aortic valve closure may approach the time of pulmonic valve closure, or even follow it, resulting in paradoxical splitting

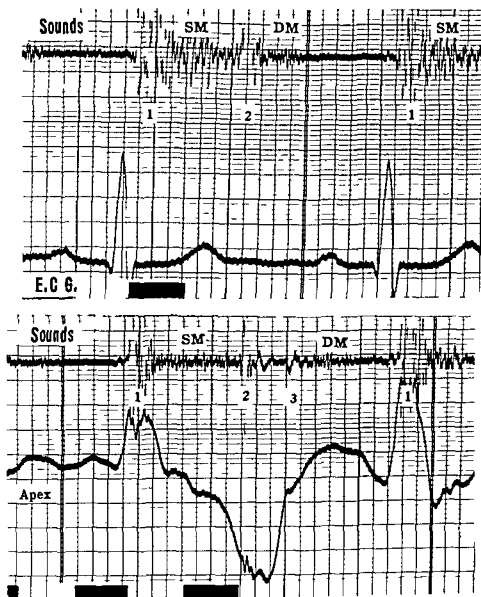


Fig 123 (Upper tracing in the pulmonic area lower tracing at the apex) Murmurs of patent ductus arteriosus associated with pulmonary artery hypertension but still having a moderate left to right shunt Note the slightly accentuated first heart sound (1) the decrescendo systolic murmur (SM) the slightly accentuated second sound (2) and the early brief decrescendo diastolic murmur (DM) In the lower tracing at the apex two additional features are seen a clear cut third heart sound (3) and in place of the early decrescendo diastolic murmur in the upper tracing there is a later mid diastolic murmur (DM) following the third sound

Third heart sound (Fig 123)

A moderate to large left to-right shunt through a patent ductus increases the flow through the left side of the heart, and a prominent third sound may result from the increased rapid early diastolic flow into the left ventricle

Gallop sounds

In patients who have congestive heart failure due to the increased work load on the left ventricle and left atrium, a protodiastolic or presystolic gallop may be noted Because of the masking effect of the loud continuous murmur, it may be impossible to distinguish atrial sounds on auscultation even though they may be recorded on the phonocardiogram Protodiastolic gallop sounds, being heard best at the apex, are more easily distinguished on routine auscultation

Continuous murmurs (Fig 124)

The typical continuous machinery murmur encountered in patients with patent ductus arteriosus varies greatly in intensity, frequency, and quality The murmur is crescendo until the timing of the second heart sound, which it frequently obliterates it then continues in a decrescendo fashion throughout diastole Sometimes it is high in frequency and almost musical At other times it is of lower frequency and comparatively rough, the unevenness of the systolic murmur is particularly characteristic

The murmur is loudest at the second left interspace and under the left clavicle but in some cases, especially in infants it may be heard best at the third or fourth interspace at the left sternal border, or even at the apex consequently, it may be confused with the murmur of ventricular septal defect

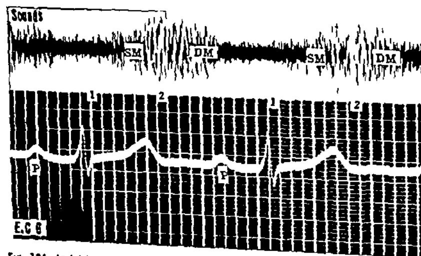


Fig 124 (2 L I S Log) High frequency high intensity crescendo decrescendo continuous murmur with no clear cut first or second heart sound visible

It is necessary to explain the accentuation of the murmur late in systole and early in diastole, whereas the greatest pressure difference between the two arterial systems occurs in mid systole The most likely explanation is that the murmur results not from flow through the ductus itself, but from flow out of the relatively narrow ductus into the dilated pulmonary artery The time taken for the pulse wave to reach the orifice of the duct and the added resistance to flow in the duct itself may account for the "shift to the right" of the murmur

If exceedingly loud, the murmur may be transmitted well over the entire upper part of the chest, in the neck, and through to the back Often it is accompanied by a late systolic early diastolic thrill coinciding with the maximal intensity of the murmur The great variability in ductus murmurs seems to depend on two factors (1) the size of the ductus, and (2) the degree of the pulmonary vascular resistance (see pp 248, 249)

SMALL SHUNTS WITHOUT PULMONARY VASCULAR OBSTRUCTION Patients *with small ducts have typical continuous machinery murmurs, frequently* with a rather low intensity and a medium to high pitch

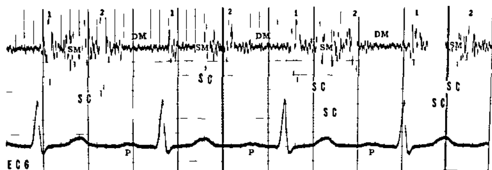


Fig 125 (Apex Log) Multiple systolic clicks occurring with patent ductus arteriosus *Note the variable intensity fairly loud first sound (1) 0 07 sec after the onset of the QRS* complex the somewhat diamond shaped systolic murmur (SM) with multiple loud systolic clicks (SC) the accentuated second heart sound (2) and the low frequency diastolic murmur (DM)

LARGE SHUNTS WITHOUT PULMONARY VASCULAR OBSTRUCTION (FIG 125) The continuous murmur in a patient who has a large patent ductus is usually grade 4 or louder and has a striking machinery *quality, often with loud* clicking sounds during systole and early diastole It would be possible, but extremely rare, for such a patient not to have a murmur if the ductus were of sufficient size and anatomically so positioned that no turbulence resulted This would be similar to the anatomic arrangements encountered in some types of true truncus arteriosus

EFFECT OF RESPIRATION ON THE CONTINUOUS MURMUR Respiration may alter considerably the systolic or the diastolic components of the continuous murmur Franck[4] pointed out that the systolic component may be accentuated by inspiration, since this results in a fall of pulmonary resistance and

an increased flow across the ductus The diastolic component is similarly, but less strikingly, affected During expiration the pulmonary resistance is somewhat increased, and the continuous murmur diminishes in intensity The extreme of this condition is shown by the Valsalva maneuver during which the continuous murmur may disappear completely, and the second heart sound, which may have been inaudible previously, may become obvious

CONTINUOUS MURMURS DUE TO CAUSES OTHER THAN PATENT DUCTUS ARTERIOSUS Continuous murmurs last throughout the entire cardiac cycle, usually they are crescendo decrescendo in configuration, and they may or may not have a machinery quality Commonly they represent extracardiac lesions and arise in collateral vessels or in the great veins from communications between systemic arteries and veins (Fig 126) or systemic and pulmonary arteries or from constrictions in the aorta or pulmonary arteries (Fig 127)

Venous hum (Fig 128) One of the most common differential diagnostic problems in young children who have continuous murmurs is the venous hum Usually venous hums are loudest on the right side of the neck or under the right clavicle, but frequently they can be heard equally well on the left side The murmur is usually grade 2 or less in intensity, but occasionally it is grade 3+ It consists of medium to high frequency vibrations and is thought to be due to blood flow through the great veins in the neck via the jugular bulb and into the superior vena cava The intensity of the venous hum may be increased by having the patient sit up, turn or extend the neck, and it may greatly diminish or disappear if the neck is flexed or the patient lies down Occasionally, the diastolic component of the hum is more prominent than the systolic, and aortic insufficiency may be suspected As a note of caution it should be added that although frequently a venous hum is

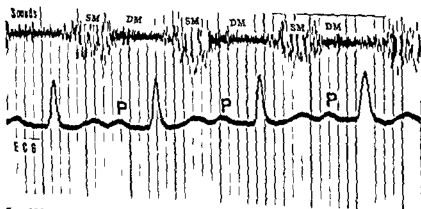


Fig 126 (Log) Continuous hemic murmur recorded over the thyroid gland in a patient who had thyrotoxicosis Note the systolic (SM) and diastolic (DM) components of a typical hemic murmur The murmur is of medium frequency and is louder during the systolic than the diastolic phase

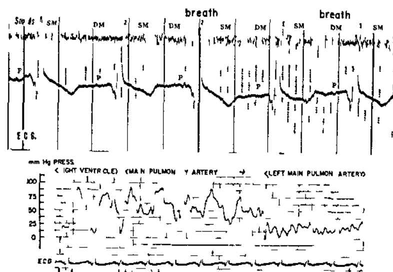


Fig 127 (Upper tracing 3 L I S Log) Phonocardiogram and cardiac catheterization pressure tracings in a patient with an atrial septal defect and partial anomalous pulmonary venous drainage This patient had a physiologic constriction in the left main pulmonary artery Repeated passage of the catheter from the main pulmonary artery into the left main pulmonary artery showed a considerable drop in pressure on each occasion On the pressure tracing a gradient from 80/50 mm of mercury to 25/10 mm of mercury persists across the constriction throughout both systole and diastole allowing flow to continue throughout the cardiac cycle The first sound (I) is of normal intensity and occurs 0 07 sec after the onset of the QRS complex The second sound is not identifiable Note the continuous systolic mur mur (SM) and diastolic murmur (DM) of medium frequency and moderate intensity This murmur is fairly constant in configuration from cycle to cycle and the superadded high frequency components are due to breath sounds as labeled

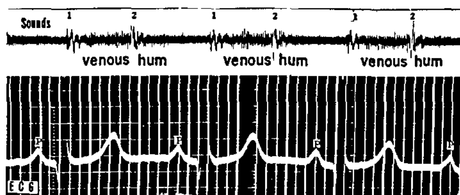


Fig 128 (2 R I S Log) Venous hum showing the continuous systolic and diastolic phase of the murmur This murmur could be obliterated by having the child lie flat or by obstructing venous return from the great veins in the neck This type of murmur can be detected in the majority of infants and children if they are examined in a sitting position Note the normal first (I) and second (2) sounds and the continuous systolic diastolic phases of the murmur which is louder in systole than in diastole

present in asymptomic children who have normal electrocardiograms, normal x rays, clear heart sounds, and no other murmurs, it may be heard especially well in certain anomalies of venous return, such as complete transposition of the pulmonary veins

Shunt operations (Fig 129) The Blalock or Potts shunt operations are performed to increase pulmonary blood flow in lesions such as tetralogy of Fallot or tricuspid atresia Successful surgery results in continuous murmurs originating from the "artificial ductus arteriosus" The intensity of the murmur varies with the size of the shunt and is loudest either to the right or the left of the upper part of the sternum, depending on which side the operation was performed The natural history of these murmurs is the same as that of the murmurs of patent ductus arteriosus, and they show a similar response to respiration In addition to the continuous murmurs, large shunts may be accompanied by apical diastolic murmurs due to the increased flow through the mitral valve

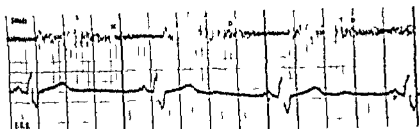


Fig 129 (2 L I S Log) Continuous murmur over the left upper part of the chest following a shunt operation for tricuspid atresia Note the normal intensity first sound (1) occurring 0 10 sec after the onset of the QRS complex of the electrocardiogram the moderate intensity second sound (2) difficult to distinguish from the murmur and the continuous murmur of medium frequency with a high intensity systolic component (SM) and a moderate intensity diastolic component (DM) This murmur increases in intensity as far as the second sound and then continues in a decrescendo manner throughout diastole

Collateral vessels If severe enough lesions causing obstruction to the normal blood flow to the lungs result in the development of collateral bronchial arteries, giving rise to continuous murmurs Usually these murmurs are of low intensity, they may be heard over the front and the back of the chest and in the axillas Because of their low intensity, careful listening while the patient s breath is held in moderate inspiration or expiration, may be necessary for their detection Bronchial collaterals may be seen in patients with severe tetralogy of Fallot, pulmonary atresia, and, occasionally even with tricuspid atresia They do not occur in pure pulmonic stenosis with intact ventricular septum Coarctation of the aorta may be accompanied by extensive collaterals to the parts of the body below the level of the coarctation These collaterals may give rise to continuous murmurs over the intercostal arteries occasionally a murmur indistinguishable from that of a patent ductus arteriosus may be heard over the internal mammary arteries

Constriction of large arteries Coarctation of the aorta or stenosis of one of the main branches of the pulmonary artery may give rise to a continuous murmur The cause of this murmur is the presence of a pressure gradient across the constricted area throughout both systole and diastole,

giving rise to a continuous flow throughout the entire cardiac cycle If sufficient turbulence results, a continuous murmur may be heard

Arteriovenous fistulas (Figs 130 and 131) Arteriovenous fistulas, whether congenital or acquired, give rise to essentially similar murmurs, namely, a continuous murmur with a crescendo decrescendo configuraton, the intensity of which varies with the size of the shunt through the fistula Often there is an accompanying thrill The diagnosis can be made easily if the fistula is at a peripheral part of the circulation, particularly when the arterial supply to the fistula is compressible and the murmur may be obliterated by simple digital pressure If the fistula is in the chest, especially between a coronary artery and a vein, or in the left upper lung, the murmur may be indistinguishable from that of patent ductus arteriosus Any arteriovenous fistula with a large shunt and a large cardiac output may bring into prominence cardiac lesions which otherwise are not significant For instance, one of our patients had a grade 4 pulmonic systolic murmur and a continuous murmur over the right kidney On removal of an arteriovenous fistula between the

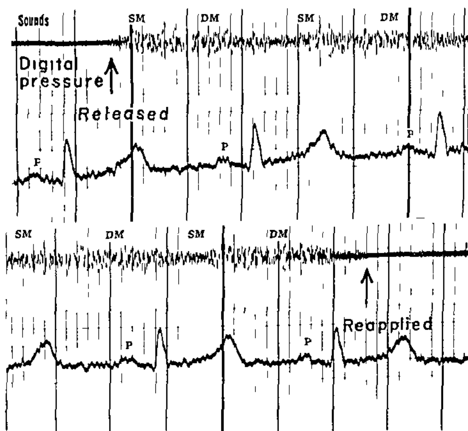


Fig 130 (Log) Continuous murmur and thrill accompanying a circoid aneurysm which was clearly visible on the forehead Firm pressure over the artery leading into the aneurysm obliterated the murmur and thrill completely Note the continuous murmur (SM DM) of *medium high frequency* and moderate intensity commencing almost immediately after the release of digital pressure over the artery leading into the aneurysm and disappearing as pressure was re applied

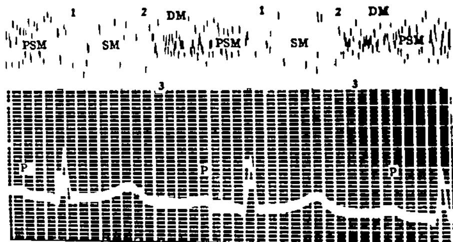


Fig 131 (L.L.S.B. Log.) Continuous murmur in a patient with an arteriovenous aneurysm involving the left coronary artery. The lesion was confirmed by exploratory thoracotomy in an 18 year old boy who was in excellent health. The first (1), second (2) and third (3) heart sounds are almost completely enveloped in the murmurs. Note the systolic murmur (SM) of high frequency and high intensity and the intense medium frequency diastolic murmur (DM) lasting throughout diastole and ending with a presystolic crescendo (PSM).

right renal artery and vein, the grade 4 pulmonic systolic murmur was reduced to grade 1, and the electrocardiogram, which had shown prominent P waves prior to operation, returned to normal. Apparently this patient had subclinical pulmonic stenosis which was made evident by the increased cardiac output through the pulmonic valve.

Truncus arteriosus (Fig 132) This anomaly consists of a common arterial trunk associated with a defect in, or rarely with complete absence

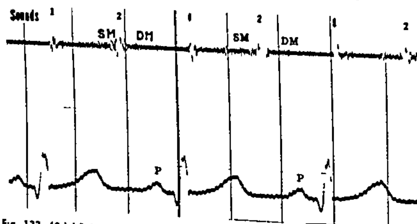


Fig 132 (2 L.I.S. Log.) Continuous murmur of low intensity and medium to high frequency in a 2 month old baby with true truncus arteriosus proved at surgery. Note the low intensity first sound (1) 0.09 sec after the onset of the QRS complex, the normal intensity second sound (2) and the low intensity murmur (SM DM) commencing during the early part of systole, crescendo up to the second sound and decrescendo during diastole. Clinically this murmur sounds to and fro rather than continuous.

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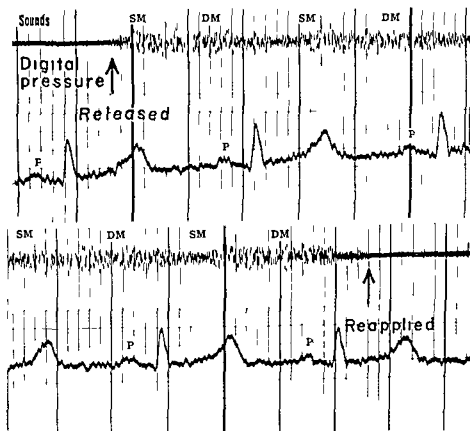


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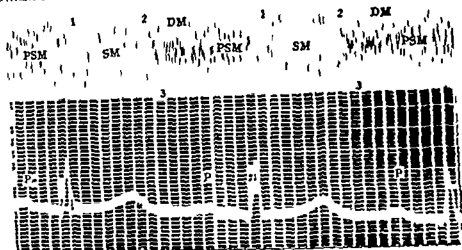


Fig 131 (L.L.S.B Log) Continuous murmur in a patient with an arteriovenous aneurysm involving the left coronary artery The lesion was confirmed by exploratory thoracotomy in an 18 year old boy who was in excellent health The first (1) second (2) and third (3) heart sounds are almost completely enveloped in the murmurs Note the systolic murmur (SM) of high frequency and high intensity and the intense medium frequency diastolic murmur (DM) lasting throughout diastole and ending with a presystolic crescendo (PSM)

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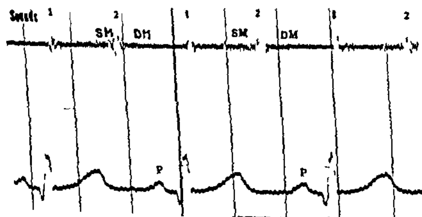


Fig 132 (2 L.I.S Log) Continuous murmur of low intensity and medium to high frequency in 2 month old baby with *true truncus arteriosus* proved at surgery Note the low intensity first sound (1) 0 09 sec *after the onset of the QRS complex* the normal intensity second sound (2) and the low intensity murmur (*SM DM*) commencing during the early part of systole crescendo up to the *second sound* and decrescendo during diastole Clinically this murmur sounded to and fro rather than continuous

of, the ventricular septum The first sound is normal or slightly accentuated Frequently, the second sound is loud, and, occasionally, it is markedly accentuated It would be expected that the second sound would be single, but for reasons which are not clear, this is not always the case

Continuous murmurs, which may be accompanied by a thrill, are present if the patient has a good pulmonary flow and, thus, little if any cyanosis The continuous murmur is similar to that of a patent ductus arteriosus It may be loudest to the left or the right of the sternum, and the intensity varies with respiration Apical mid diastolic or presystolic murmurs may be present also

Ruptured sinus of Valsalva (Fig 133) Rupture of the sinus of Valsalva may occur into the left ventricle, the right ventricle, the right atrium, or, very rarely, even into the left atrium If rupture occurs into the left ventricle, a loud murmur of aortic insufficiency results, whereas, if rupture occurs into the right ventricle or right atrium, a loud continuous machinery murmur results The configuration of this murmur is identical with that of the murmur of a large patent ductus arteriosus It is heard best about the middle of the left sternal border, is usually grade 5 to 6 in intensity, and may be accompanied by a thrill An apical mid diastolic rumble secondary to the increased flow through the mitral valve may be noted also

Medial necrosis of the aorta Cystic degeneration of the media of the aorta may be accompanied by the formation of a dissecting aneurysm with considerable dilatation of the ascending aorta and the aortic valve ring This may result in marked aortic insufficiency, with aortic systolic and

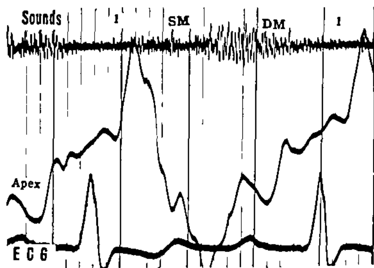


Fig 133 (Recorded at 3 L I S Log with the volume control on the phonocardiogram turned down to an amplitude of two instead of the usual six on this machine because of the intensity of the murmur) Continuous murmur with accentuation at the expected time of the second sound in a 28 year old patient with a ruptured sinus of Valsalva Note the extremely low intensity *first sound (1) due to the volume control being turned down* and the systolic (SM) and diastolic (DM) components of the murmur The second sound cannot be identified Clinically this murmur was grade 6

diastolic murmurs which are actually to and fro rather than continuous, but the differentiation may be difficult. This lesion should be suspected in any patient who has Marfan's syndrome with a to and fro or a continuous murmur over the aortic area.

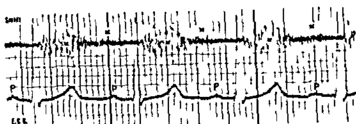


Fig 134 (2 R I S Log) A to and fro murmur in a patient with a left to right shunt through a ventricular septal defect and aortic regurgitation due to a defective cusp of the aortic valve. Note the poorly identified first (1) and second (2) sounds, the diamond shaped systolic murmur (SM) of medium frequency and moderate to high intensity, and the decrescendo diastolic murmur (DM) of medium frequency and moderate intensity.

Rupture of an aortic aneurysm into the pulmonary artery (Fig 134) This condition resembles rupture of a sinus of Valsalva, but usually occurs in patients in the older age groups. The sudden onset of a continuous murmur in a patient who has a thoracic aortic aneurysm or a history of lues may suggest the diagnosis.

Respiratory artefact (Fig 135) Respiratory artefact, especially in chil

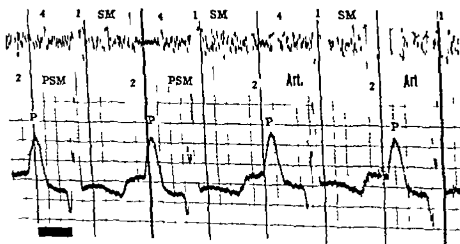


Fig 135 (2 L I S Log) An apparently continuous murmur due to respiratory artefact in the last half of a recording made over the pulmonary area in a patient with severe valvular pulmonic stenosis and a right to left shunt at the atrial level. Note the normal first sound (1) 0.06 sec after the onset of the QRS complex, the diamond shaped systolic murmur (SM) of medium frequency and moderate intensity, the narrow second sound (2) of normal intensity, the prominent atrial sound (4) 0.07 sec after the *onset of the P* wave, and the presystolic murmur (PSM) of low to moderate intensity. In the last half of the tracing respiratory sounds labeled Art for artefact give the phonocardiographic appearance of a continuous murmur.

dren with rapid respirations, may simulate a continuous murmur on the phonocardiogram In this respect the phonocardiogram is inferior to human hearing in the interpretation of murmurs Whereas the machine records faithfully all sounds which are produced, the human hearing mechanism, by virture of experience, is able to reject all extraneous sounds

APICAL DIASTOLIC MURMURS (FIGS 136 AND 137)

With moderate or large shunts through a patent ductus arteriosus, there is increased flow through the mitral valve As a result an apical diastolic

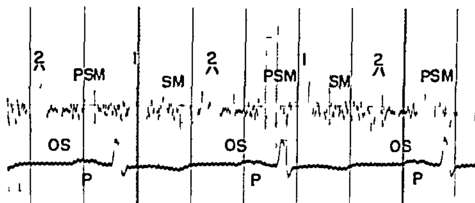


Fig 136 (Apex Log) Mid diastolic and presystolic murmurs in patient with patent ductus arteriosus Note the high intensity first sound (1) 0 08 sec after the onset of the Q wave the holosystolic murmur (SM) of medium frequency and moderate intensity and varying between crescendo decrescendo and plateau shaped the well split (0 03 to 0 04 sec) second sound (2) of moderate intensity the narrow sound (OS) of high frequency and high intensity presumably an opening snap 0 06 sec after the termination of the second sound and the late diastolic presystolic murmur (PSM) of medium frequency and fairly high intensity with out presystolic crescendo

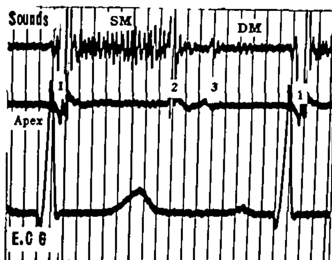


Fig 137 (Apex Log) Apical third sound and mid diastolic murmur in patent ductus arteriosus Note the normal first sound (1) 0 06 sec after the onset of the QRS complex the holosystolic late crescendo systolic murmur (SM) of moderate intensity and medium frequency the normal second sound (2) the low intensity third sound (3) and the mid diastolic murmur (DM) of low frequency and low intensity

murmur of low to medium frequency, and of low to moderate intensity usually can be heard The murmur occurs in mid-diastole, starting at the time of the third heart sound and frequently continuing into presystole

This murmur is loudest with large left to right shunts and is less loud or absent when the shunt is smaller, either because of a small ductus or because of pulmonary vascular obstruction

BASAL DIASTOLIC MURMURS (FIG 138)

A diastolic murmur alone is never a sign of patent ductus arteriosus Murmurs of aortic or pulmonary insufficiency always indicate additional lesions such as aortic valve anomalies, pulmonary vascular obstruction or, perhaps a greatly dilated pulmonary artery with pulmonary valve insufficiency In patients who have severe pulmonary vascular obstruction and an accentuated pulmonic second sound but do not have a murmur when resting it is possible that the murmur of pulmonary insufficiency may develop with exercise This would be an exceptional situation and would in no way suggest the specific diagnosis of patent ductus arteriosus

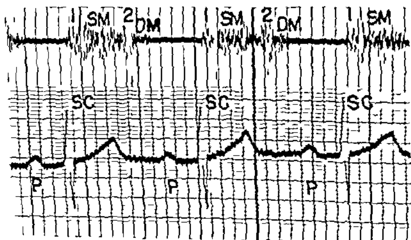


Fig 138 (2 L I S Log) Accentuated pulmonic second sound and early systolic click together with a systolic and an early diastolic murmur in patient with patent ductus arteriosus with pulmonary vascular obstruction Note the first sound (1) of moderately low intensity 0 05 to 0 06 sec after the onset of the Q wave the high intensity early systolic click (SC) 0 08 to 0 09 sec after the onset of the Q wave the holosystolic murmur (SM) of medium to high frequency moderate intensity and variable configuration the high intensity second sound (2) and the early diastolic short high frequency decrescendo diastolic murmur (DM) Neither by auscultation nor by phonocardiography can one be certain whether this murmur represents pulmonary regurgitation or is the diastolic component of the shunt murmur

MURMURS OF PATENT DUCTUS ARTERIOSUS WITH PULMONIC OR AORTIC STENOSIS (FIG 139)

In some patients with patent ductus arteriosus a stenotic systolic murmur may be heard at the second left or second right interspace which may be transmitted well to the neck and back Usually these patients have true or

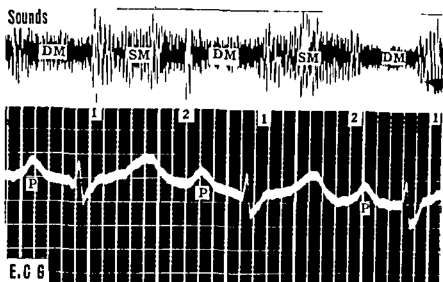


Fig 139 (2 L I S Log) Second sound varying in intensity from cycle to cycle in a patient with patent ductus arteriosus The first cycle shows a crescendo descrescendo systolic murmur (SM) with an obvious second sound (2) and a plateau shaped high frequency diastolic murmur (DM) The second cycle shows a similarly shaped systolic and diastolic murmur (SM) and (DM) but no clear cut second sound Note the normal intensity first sound (1) 0 07 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) of medium frequency and moderate to high intensity consistent with a diagnosis of pulmonic stenosis the variable intensity second sound (2) and the plateau shaped high frequency diastolic *murmur (DM)*

ganic pulmonic or aortic stenosis in addition to the patent ductus arteriosus True organic pulmonic stenosis should be suspected in those patients who have a history of maternal rubella during the first trimester of pregnancy

Effects of pulmonary vascular obstruction on the sounds and murmurs of patent ductus arteriosus (Figs 140, 141, and 142)

Pulmonary vascular obstruction is sometimes encountered in patients who have patent ductus arteriosus If it is progressive, it can cause marked

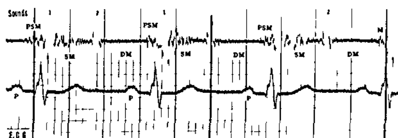


Fig 140 (Apex Log) Presystolic murmur (PSM) and an accentuated second sound (2) occurring in a patient who had pulmonary vascular obstruction with a *reversed shunt* through a patent ductus arteriosus Note the loud first heart sound (1) with the maximum vibrations 0 09 sec after the onset of the QRS complex the decrescendo systolic murmur (SM) of medium frequency and moderate intensity the loud second heart sound (2) not obviously split the low frequency low intensity diastolic vibrations (DM) and the short crescendo presystolic murmur (PSM) commencing 0 08 sec after the beginning of the P wave of the electrocardiogram

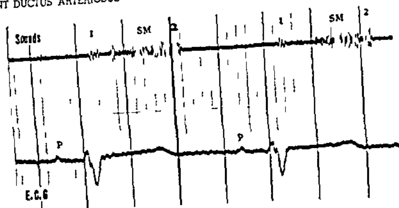


Fig 141 (2 L I S Log) Crescendic systolic murmur in patent ductus arteriosus with a small left to right shunt systemic pressures in the pulmonary artery and markedly increased pulmonary vascular resistance Note the low intensity first sound (1) 0 08 sec after the onset of the Q wave the systolic murmur (SM) commencing 0 08 sec after the first sound and crescendic up to the second sound and the normal intensity second sound (2) split by 0 04 sec

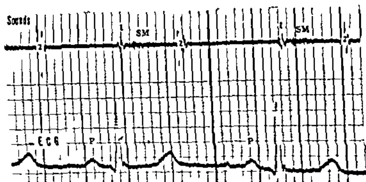


Fig 142 (2 L I S Log) Normal first sound and slight accentuation of the pulmonic second sound without significant murmurs in a patient who had patent ductus arteriosus with severe pulmonary artery hypertension This record taken at the second left interspace is remarkable in that it is almost entirely normal It was the slight accentuation of the second sound with a minor degree of abnormal right ventricular hypertrophy which led us to investigate The catheter passed freely through a large patent ductus The patient succumbed three days after operation Note the low intensity first heart sound (1) the minimal systolic murmur (SM) and the slightly accentuated second sound (2)

changes in the typical continuous murmur and in the intensity of the second heart sound As the pulmonary vascular resistance increases, the pulmonary artery pressure rises, and the gradient from aorta to pulmonary artery across the ductus diminishes The final result of this is equal aortic and pulmonary artery pressures with a minimal left to right or right to left shunt and no significant murmur Various stages in the development of pulmonary vascular obstruction may cause the following alterations in auscultatory phenomena 1 The diastolic component of the continuous murmur may lessen and finally disappear as the pulmonary diastolic pressure rises and diminishes the diastolic gradient across the ductus 2 The pulmonary second

sound increases in intensity as the pressure causing pulmonary valve closure rises 3 The apical diastolic murmur disappears as the left to right shunt diminishes and the flow through the left side of the heart and across the mitral valve decreases 4 Finally, as the systolic pressure in the pulmonary artery reaches that in the aorta, the systolic gradient across the ductus diminishes, and the systolic murmur also disappears

Effect of surgery on the murmurs of patent ductus arteriosus

Successful repair of a patent ductus arteriosus results in complete disappearance of the continuous murmur and the apical diastolic murmur

A grade 1 or grade 2 systolic murmur over the pulmonic or aortic area may persist for a few days or weeks after operation An apical systolic murmur representing functional mitral regurgitation may persist for a year or more But, assuming that no other lesion is present, this is one instance where successful surgery should be followed eventually by a return to perfectly normal cardiovascular sounds and murmurs

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19 / Ventricular Septal Defects

Ventricular septal defects vary considerably in size, as a result, there is considerable variation in the associated left to right shunt and the accompanying murmurs

It is well known that loud systolic murmurs are often associated with small ventricular defects of minor physiologic importance while the larger, more serious defects may be associated with less prominent systolic murmurs

Small left to right shunts (Figs 143 and 144)

Heart sounds and murmurs The first heart sound is normal in splitting and intensity and is heard best at the apex or lower left sternal border

The second sound is moderately split and is normal to 1+ in intensity Usually a third sound is heard at the apex

The systolic murmur is the striking feature of the examination It is loud grade 3 to 5 or even 6, in intensity, and, almost without exception it is accompanied by a thrill The murmur is heard best at the mid or lower left sternal border and is transmitted moderately well in a radial manner

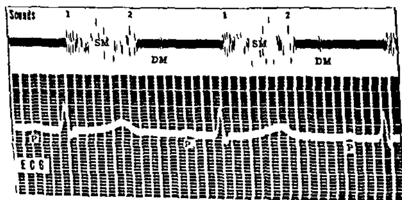


Fig 143 (LLSB Log) Short low intensity vibrations around the time of the normal third sound in a patient with small ventricular septal defect Note the normal first (1) and second (2) heart sounds, the holosystolic murmur (SM) of high intensity and medium frequency and the short diastolic vibrations (DM) of low frequency and low intensity lasting 0 04 sec It is not possible to differentiate between a murmur and a third sound under these circumstances In the first complex the vibrations are regular are of medium frequency and resemble a murmur In the second complex they are irregular and look more like a third sound

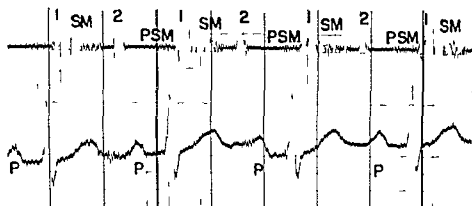


Fig 144 (L L S B Log) Holosystolic decrescendo systolic murmur with a short presystolic murmur in a patient with a ventricular septal defect Note the first sound (1) of normal intensity 0 06 to 0 07 sec after the onset of the Q wave the decrescendo systolic murmur (SM) of high frequency and moderate intensity continuing throughout almost all of systole the narrow moderate intensity second sound (2) and the presystolic murmur (PSM) of low intensity and medium frequency without presystolic crescendo

Phonocardiograms reveal the murmur to be plateau shaped, continuing from the first sound through to the second Occasionally it may be decrescendo or may show a diamond configuration due to blood being shunted at high pressure through a small orifice

It may be extremely difficult and sometimes quite impossible to differentiate this murmur from that of infundibular pulmonic stenosis The murmur may be harsh in both instances, and, although murmurs of pulmonic stenosis are transmitted better to the neck, a loud murmur from a ventricular defect may also be heard in the neck Since the left to right shunt is relatively small *in this type of defect, a mid diastolic apical murmur usually is not present*

MODERATE TO LARGE LEFT-TO RIGHT SHUNTS
(FIGS 145, 146, 147, AND 148)

HEART SOUNDS AND MURMURS The first sound is usually normal or 1+ *and is loudest at the apex* or lower left sternal border The second sound is moderately well split at the pulmonic area and pulmonary closure may be accentuated A third sound is heard clearly at the apex A holosystolic plateau shaped grade 3 to 4 murmur, together with a thrill is present at the lower or mid left sternal border, in addition, there is a grade 2 to 3 apical diastolic rumble following the third heart sound The diastolic murmur is low to medium pitched and often continues into presystole, rarely with presystolic crescendo it is probably caused by the greatly increased diastolic flow through the mitral valve

DIFFERENTIAL DIAGNOSIS A ventricular septal defect with a large left to right shunt may be confused with a large patent ductus, especially when a moderate degree of pulmonary vascular obstruction is present The murmurs may be identical and in a large patent ductus a systolic thrill is sometimes

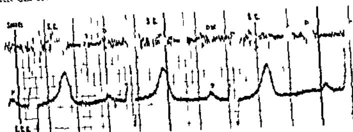


Fig 145 (Apex Log) Loud systolic murmur and a loud long diastolic murmur in a patient having a ventricular septal defect with a large left to right shunt Note the loud first heart sound (1) with the maximal valvular component 0 08 sec after the Q wave of the electrocardiogram the loud systolic click (SC) at the end of the first third of systole 0 18 sec after the onset of the QRS complex the crescendo decrescendo systolic murmur (SM) of medium frequency and moderate intensity the loud second sound (2) and the loud diastolic murmur (DM) of medium frequency in middle and late portions of diastole

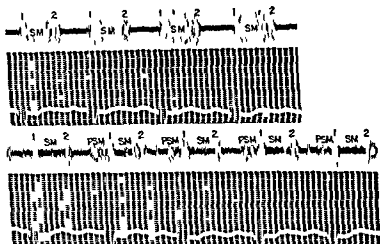


Fig 146 Loud systolic murmur at the left sternal border and a prominent apical presystolic murmur associated with a large left to right shunt (pulmonary blood flow was four times systemic flow) in ventricular septal defect

Upper tracing (3 L I S Log) Note the first sound (1) of normal intensity which is difficult to identify clearly as it continues straight into the high intensity systolic murmur (SM) the plateau type of systolic murmur (SM) of high intensity and high frequency occupying all of systole and the narrowly split (0 02 sec) second sound (2) of normal intensity

Lower tracing (Apex Log) Note the accentuated first sound (1) 0 07 sec after the onset of the Q wave which is clearly distinguishable from the systolic murmur the holosystolic murmur (SM) of high frequency and moderate intensity the narrowly split second sound (2) of normal intensity and the presystolic murmur (PSM) of high intensity and medium to high frequency

felt over the lower anterior part of the chest It is best to realize that occasionally it is impossible to distinguish a large left to right shunt through a ventricular septal defect from a large shunt through a patent ductus arteriosus by x ray by electrocardiograms, or by clinical examination This is especially true in very young patients

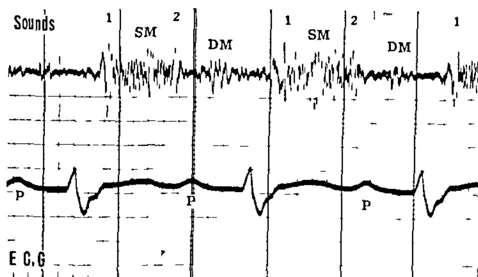


Fig 147 (Apex Log) Apical mid diastolic murmur in a patient with a moderate sized ventricular septal defect Note the normal intensity first sound (1) with the major component 0 10 sec after the Q wave of the electrocardiogram the holosystolic murmur (SM) of medium frequency and moderate intensity the normal intensity second sound (2) the mid diastolic murmur (DM) of low frequency and moderate intensity and the first degree heart block The P R interval equals 0 18 sec with a rate of 125 beats per minute

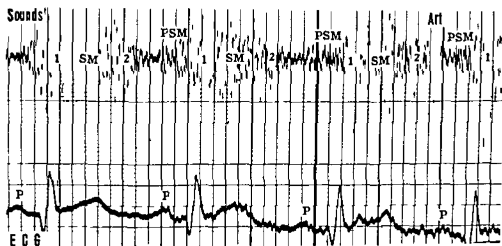


Fig 148 (Apex Log) Very loud systolic murmur with a presystolic crescendo murmur in an infant with ventricular septal defect with a large left to right shunt and congestive failure Note the normal first sound (1) 0 08 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) of medium frequency and high intensity the normal second sound (2) and the moderate intensity crescendo presystolic murmur (PSM)

Ventricular septal defect with pulmonic stenosis

In some children with ventricular defects and left to right shunts significant valvular or infundibular pulmonic stenosis develops, this may occur at any age, ranging from a few months to the early teens If the degree of the pulmonic stenosis increases sufficiently, it may become the dominant lesion, and, on exercise, the shunt may reverse, becoming right to left, with

the development of cyanosis Hence, the patient may change from having a ventricular septal defect with a left to right shunt, increased pulmonary flow, and mild pulmonary stenosis to having true tetralogy of Fallot with a right to left shunt and diminished pulmonary flow Under these circumstances certain changes occur in the typical heart sounds and murmurs of ventricular septal defect

The pulmonic second sound becomes diminished As the left to right shunt lessens, the apical diastolic rumble disappears With the development of valvular or infundibular stenosis, a stenotic systolic murmur can be heard at the second third, or fourth left interspace, this is conducted well to the neck and back When the pulmonic stenosis is infundibular there may be little apparent change in the systolic murmur at the lower left sternal border

VENTRICULAR SEPTAL DEFECT WITH PULMONARY VASCULAR OBSTRUCTION (FIGS 149, 150 AND 151)

Increased pulmonary vascular resistance may be present with a ventricular defect, resulting in pulmonary artery hypertension Because of the increased pressure in the pulmonary circuit, the pulmonic second sound becomes loud Because of the small left to right shunt in these patients, an apical diastolic rumble may not be present This is Eisenmenger's syndrome, that is a ventricular septal defect with pulmonary vascular obstruction and, finally reversal of the shunt, with the development of so called cyanose tardive"

Fully developed severe pulmonary vascular obstruction may be accompanied by severe pulmonic regurgitation with the dominant murmur being a loud decrescendo insufficiency blow following an accentuated pulmonic

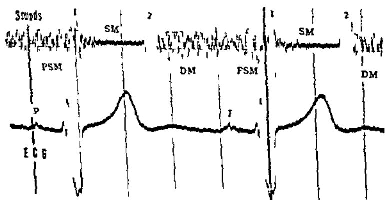


Fig 149 (2 L I.S Log) Pulmonary insufficiency murmur with a presystolic crescendo in a patient with ventricular septal defect and pulmonary vascular obstruction (Eisenmenger's syndrome) Note the normal first sound (1) 0 08 sec after the onset of the QRS complex the low intensity systolic murmur (SM) the greatly accentuated second sound (2) and the diastolic murmur (DM) of high frequency and moderately high intensity commencing immediately after the second sound and continuing throughout diastole to terminate in a presystolic crescendo (PSM)

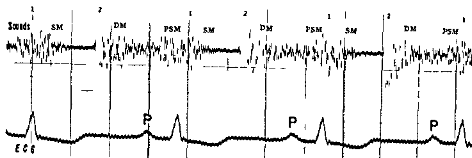


Fig 150 (2 L I S Log) Pulmonary insufficiency murmur with presystolic crescendo in a patient with ventricular septal defect and pulmonary vascular obstruction (Eisenmenger's syndrome) Note the normal first sound (1) This sound is labeled at slightly different time intervals in relation to the QRS complex in each cycle emphasizing the difficulty in being certain just which group of vibrations constitutes the first sound Note also the insignificant systolic murmur (SM) *the accentuated second sound (2) and the diastolic murmur (DM) of* high frequency and high intensity which is decrescendo following the second sound and then terminates in a presystolic crescendo (PSM)

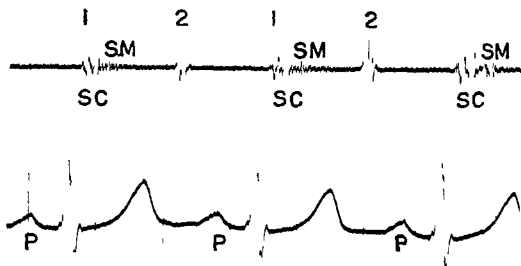


Fig 151 (2 L I S Log) Early systolic ejection sound and accentuated pulmonary second sound but no significant murmurs in patient with ventricular septal defect and pulmonary vascular obstruction Note the first sound (1) of moderate to low intensity 0 08 sec after *the onset of the Q wave the early systolic click (SC) 0 12 sec after* the onset of the Q wave and of slightly greater intensity than the first sound the vibrations occupying the early part of systole (SM) which are of low intensity and medium to high frequency and the narrow high intensity second sound (2)

second sound Occasionally this murmur may terminate in a presystolic crescendo comparable with the Austin Flint presystolic murmur of aortic regurgitation The exact cause of this presystolic accentuation is not certain

VENTRICULAR SEPTAL DEFECT WITH AORTIC REGURGITATION (FIGS 152, 153, AND 154)

This condition is important in that it may be mistaken for a patent ductus arteriosus It consists of a defect in the membranous ventricular septum associated with a defective or distorted aortic valve cusp

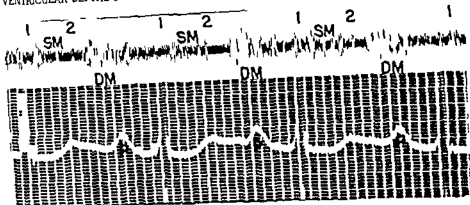


Fig 152 (4 LIS Log) Systolic and diastolic murmurs without a clearly distinguishable second heart sound in patient with ventricular septal defect and aortic regurgitation. Neither the first sound (1) nor the second sound (2) is clearly visible. Note the holosystolic plateau shaped systolic murmur (SM) of medium frequency and moderate intensity and the high frequency diastolic murmur (DM) with a crescendo decrescendo configuration early in diastole gradually diminishing throughout the rest of diastole.

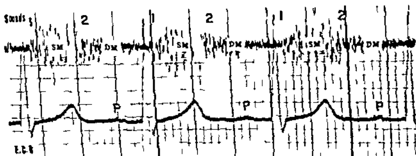


Fig 153 (3 LIS Log) Crescendo decrescendo systolic murmur with a long diastolic decrescendo phase in patient with ventricular septal defect and aortic insufficiency following bacterial endocarditis. In this area the murmur sounded continuous with a machinery quality. Neither the first sound (1) nor the second heart sound (2) is clearly identifiable. Note the high frequency high intensity systolic murmur (SM) with a diamond configuration and the diastolic murmur (DM) of high frequency and moderate intensity which is decrescendo in configuration and lasts throughout diastole.

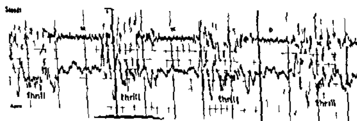


Fig 154 (Apex Log with apex cardiogram) Apical diastolic rumble in patient who had a ventricular septal defect with aortic insufficiency following *bacterial endocarditis*. Note the moderately loud first sound *(1)*, the diamond shaped systolic *murmur (SM)* of high frequency and high intensity, the normal intensity second sound (2) and the *diastolic* murmur (DM) of low frequency and low intensity *lasting throughout* most of diastole. This murmur may be due in part to the increased diastolic flow through the mitral valve and in part to the greatly dilated left ventricle. A systolic thrill can be seen on the apex cardiogram.

First and second heart sounds The first heart sound is moderately accentuated because of the hyperactive left ventricle The pulmonic second sound, which actually may be accentuated, nevertheless may be inaudible because of the masking effect of the loud murmurs Aortic valve closure is diminished as a consequence of the aortic insufficiency

Murmurs The associated murmurs often are extremely loud and discovered early in life They are systolic and diastolic in timing and are audible over the entire precordium,, loudest along the left sternal border Although truly to and fro in character, they may sound continuous The systolic murmur is heard best at the mid or lower left sternal border, and the diastolic murmur around the third left interspace The systolic murmur may be grade 5 to 6, and the diastolic murmur grade 2 to 5 At the upper sternal border the two murmurs may sound continuous The great intensity of both murmurs in some cases allows them to be heard clearly over the entire precordium, neck, and back Because of the increased output through the aortic valve with each systole, the systolic murmur over the aortic area may sound stenotic and may appear diamond shaped on the phonocardiogram An apical, mid diastolic rumble, secondary to the large left to right shunt, is usually present

Single ventricle

Patients with a single ventricle have their congenital heart disease diagnosed soon after birth They constitute a complex group Other congenital cardiac anomalies, including transposition of the great vessels, often with pulmonic stenosis or atresia, may be encountered, while lesions of the atrioventricular valves, especially mitral atresia, mitral insufficiency, tricuspid atresia, or tricuspid stenosis, are common

First and second heart sounds The first heart sound is of normal intensity or is slightly accentuated, it is loudest at the lower left sternal border

The second sound is variable It is single in patients who have associated aortic atresia or pulmonary atresia It is split in patients with normal outflow tracts, and pulmonary closure is accentuated if pulmonary hypertension is present Because of the frequent accompaniment of some degree of transposition, it is not safe to assume that the second sound at the pulmonary area represents pulmonic valve closure and that the second sound at the aortic area represents aortic valve closure

Murmurs Systolic murmurs at the lower left sternal border are common Both mid diastolic and presystolic murmurs occur frequently in patients who have intact atrioventricular valves and increased pulmonary flow In 2 of our patients, loud mid diastolic murmurs were noted at the lower right sternal border

In the presence of stenosis of one of the semilunar valves, a stenotic murmur may be encountered, but not necessarily at the expected site

20 / Atrial Septal Defect

Variations from normal in the heart sounds and murmurs of patients with atrial septal defect are dependent on several factors The most important of these are (1) the size and position of the defect (for example, whether secundum or primum), (2) the volume of shunt through the defect, and (3) the presence of associated lesions, such as pulmonic stenosis, pulmonary vascular obstruction, or mitral valve disease

We shall first discuss the foramen secundum atrial septal defect and later the less common foramen primum lesion

FORAMEN SECUNDUM ATRIAL DEFECT (FIGS 155 AND 156)

AUSCULTATION

FIRST HEART SOUND The first sound is not remarkable in intensity, but it may be moderately well split

EARLY SYSTOLIC CLICK In patients with a dilated main pulmonary artery, and particularly in those with pulmonary artery hypertension, an early systolic ejection sound may be clearly audible

SECOND HEART SOUND Usually, in normal children the second heart sound in the pulmonic area is split, but the interval between aortic and pulmonic closure does not exceed 0 03 sec during expiration and 0 05 sec during

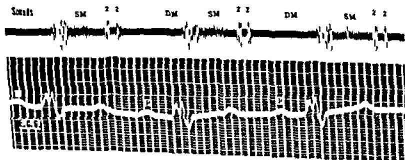


Fig 155 (2 L I S Log) Obvious splitting of the second sound in a patient with a foramen secundum atrial septal defect Note the moderate intensity broad first sound (1) 0 12 sec after the onset of the QRS complex the well split (0 06 sec) second sound (2 2) with the first component slightly more intense than the second the systolic murmur (SM) of moderate intensity medium frequency and of no specific configuration and a few low intensity late diastolic and presystolic vibrations (DM)

First and second heart sounds The first heart sound is moderately accentuated because of the hyperactive left ventricle The pulmonic second sound, which actually may be accentuated, nevertheless may be inaudible because of the masking effect of the loud murmurs Aortic valve closure is diminished as a consequence of the aortic insufficiency

Murmurs The associated murmurs often are extremely loud and discovered early in life They are systolic and diastolic in timing and are audible over the entire precordium,, *loudest along the left sternal border Although* truly to and fro in character, they may sound continuous The systolic murmur is heard best at the mid or lower left sternal border, and the diastolic murmur around the third left interspace The systolic murmur may be grade 5 to 6, and the diastolic murmur grade 2 to 5 At the upper sternal border the two murmurs may sound continuous The great intensity of both murmurs in some cases allows them to be heard clearly over the entire precordium, neck, and back Because of the increased output through the aortic valve with each systole, the systolic murmur over the aortic area may sound stenotic and may appear diamond shaped on the phonocardiogram An apical, mid diastolic rumble, secondary to the large left to right shunt, is usually present

Single ventricle

Patients with a single ventricle have their congenital heart disease diagnosed soon after birth They constitute a complex group Other congenital cardiac anomalies, including transposition of the great vessels, often with pulmonic stenosis or atresia, may be encountered, while lesions of the atrio*ventricular valves, especially mitral atresia, mitral insufficiency, tricuspid* atresia, or tricuspid stenosis, are common

First and second heart sounds The first heart sound is of normal intensity or is slightly accentuated, it is loudest at the lower left sternal border

The second sound is variable It is single in patients who have associated aortic atresia or pulmonary atresia It is split in patients with normal outflow tracts, and pulmonary closure is accentuated if pulmonary hypertension is present Because of the frequent accompaniment of some degree of transposition, it is not safe to assume that the second sound at the pulmonary area represents pulmonic valve closure and that the second sound at the aortic area represents aortic valve closure

Murmurs Systolic murmurs at the lower left sternal border are common Both mid diastolic and presystolic murmurs occur frequently in patients who have intact atrioventricular valves and increased pulmonary flow In 2 of our patients, loud mid diastolic murmurs were noted at the lower right sternal border

In the presence of stenosis of one of the semilunar valves, a stenotic murmur may be encountered, but not necessarily at the expected site

20 / Atrial Septal Defect

Variations from normal in the heart sounds and murmurs of patients with atrial septal defect are dependent on several factors The most important of these are (1) the size and position of the defect (for example whether secundum or primum), (2) the volume of shunt through the defect, and (3) the presence of associated lesions, such as pulmonic stenosis pulmonary vascular obstruction, or mitral valve disease

We shall first discuss the foramen secundum atrial septal defect and later the less common foramen primum lesion

FORAMEN SECUNDUM ATRIAL DEFECT (FIGS 155 AND 156)

AUSCULTATION

First heart sound The first sound is not remarkable in intensity, but it may be moderately well split

Early systolic click In patients with a dilated main pulmonary artery and particularly in those with pulmonary artery hypertension an early systolic ejection sound may be clearly audible

Second heart sound Usually in normal children, the second heart sound in the pulmonic area is split but the interval between aortic and pulmonic closure does not exceed 0 03 sec during expiration and 0 05 sec during

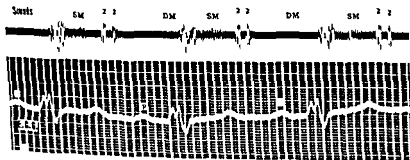


Fig 155 (2 L I S Log) Obvious splitting of the second sound in a patient with a foramen secundum atrial septal defect Note the moderate intensity broad first sound (1) 0 12 sec after the onset of the QRS complex the well split (0 06 sec) second sound (2 2) with the first component slightly more intense than the second the systolic murmur (SM) of moderate intensity medium frequency and of no specific configuration and a few low intensity late diastolic and presystolic vibrations (DM)

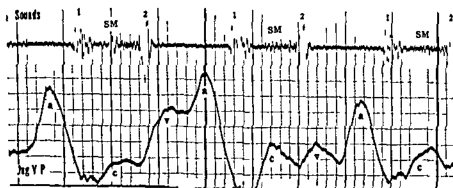


Fig 156 (Apex Log and jugular venous pulse) Jugular venous pulse tracing in a patient with an atrial septal defect demonstrating the prominent a waves followed by a sudden drop before the c wave Note the normal first sound (1) of moderate intensity split by 0 02 sec the systolic murmur (SM) of low intensity and medium frequency the moderately accentuated second sound (2) and the prominent a wave of the jugular venous pulse followed by a deep negative wave preceding the c wave

inspiration In patients with atrial septal defect, the splitting of the second sound in the pulmonic area is unusually wide and does not vary significantly with respiration The relation of aortic closure to electrical systole remains constant, thus, the wide splitting of the second sound is due to delay in the pulmonic valve closure The intensity of the two components of the split is usually approximately equal, but the pulmonic component may be accentuated if pulmonary hypertension is present Wide splitting of the pulmonic second sound is often the first clinical indication of the presence of an atrial defect, and it seems all the more striking as the associated murmurs may be of comparatively low intensity

Cause of the wide splitting It would seem that the widely split second sound at the pulmonic area in patients with atrial septal defect could be explained by the almost invariable presence of right bundle branch block However, since patients who have right bundle branch block but no atrial septal defect do not show the same consistent wide splitting of the pulmonic second sound some other explanation must be sought The most obvious cause would seem to be prolongation of right ventricular systole due to the greatly increased flow, and this is further substantiated by the relative independence of the degree of splitting from respiration The role played by increased right ventricular flow in the delayed pulmonic valve closure will be better assessed when more patients have been re examined following surgical closure of atrial defects Successful closure should result in a reduction in the degree of splitting since right bundle branch block usually does not diminish after surgery the only other obvious factor would be the return to normal of right ventricular flow, with a consequent reduction in duration of right ventricular systole (Figs 157 and 158)

Third heart sound A third heart sound may be heard clearly in patients with atrial defect, it is loudest between the apex and the lower left sternal border Because of the left to right shunt at the atrial level, there is increased

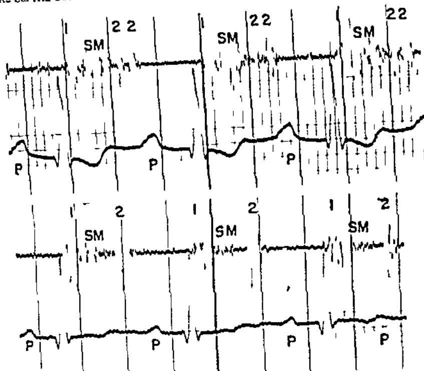


Fig 157 Narrowing of the splitting of the second sound after successful surgical closure of an atrial septal defect

Upper tracing (2 L I S Log) Before surgery the P R interval was 0.28 sec and the QRS interval was 0.08 sec Note the first sound (1) of normal intensity 0.11 sec after the onset of the Q wave the diamond shaped systolic murmur (SM) of moderate to high intensity and medium frequency and the wide split (0.06 sec) second sound (2 2) with the second or pulmonary component much louder than the aortic closure

Lower tracing (2 L I S Log) After surgery the P R interval was 0.16 sec and the QRS interval was 0.08 sec Note the normal intensity first sound (1) the greatly diminished systolic murmur (SM) and the narrowly split (0.02 to 0.03 sec) second sound (2)

blood flow into the right ventricle early in diastole, and the third sound probably arises principally in the right ventricle or the tricuspid valve mechanism

Early diastolic sound Leatham and Gray [1] reported that in some patients with atrial defects, an extra sound occurs early in diastole They describe the sound as being high pitched and snapping in quality, with its maximum intensity at the lower left sternal border and occurring anywhere from 0.03 to 0.12 sec after pulmonic valve closure

Pulmonic systolic murmurs A soft blowing systolic murmur, grade 2 to 3 in intensity and best heard at the pulmonic area, is usually audible in a patient with an atrial septal defect This murmur increases slightly with inspiration, diminishes with expiration and may be completely abolished by the Valsalva maneuver

The pulmonic systolic murmur is due principally to increased flow through



Fig 158 (Apex Log with apex linear tracing) Phonocardiogram of septum primum type of atrial septal defect Note the loud first heart sound (1) the decrescendo systolic murmur (SM) of moderately low intensity and medium frequency the moderately split (0 05 sec) second sound (2) of normal intensity the low intensity third sound (3) the mid diastolic murmur (DM) of low intensity and low frequency and the systolic thrill recorded on the apex cardiogram

the pulmonic valve but may be partly due to the frequently associated dilatation of the pulmonary artery The development of pulmonary vascular obstruction, with a reduction in pulmonic flow, results in diminution of the systolic murmur at the second left interspace, even though the size of the pulmonary artery may be further increased

Operative closure of the atrial defect results in considerable diminution, or even disappearance, of the pulmonic systolic murmur

Pulmonic stenosis may accompany an atrial septal defect, in which case the typical diamond shaped, rough systolic murmur of a stenotic lesion is heard This murmur is of course loudest at the second left interspace, and it is conducted well to the neck and back

Mid diastolic murmur (fig 159) With secundum defects, a mid diastolic murmur, commencing at the time of the third heart sound and occasionally continuing into presystole, can be heard between the apex and the lower left sternal border The murmur is medium to low in frequency and is grade 1 to 2+ in intensity It is accentuated with inspiration and dimin

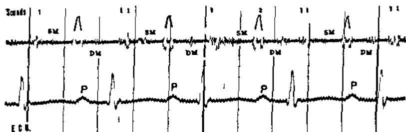


Fig 159 (Apex Log) Mid diastolic murmur in a patient with a foramen secundum atrial defect The first sound (1 1) consists of two groups of vibrations The first group is of low intensity and occurs 0 07 sec after the onset of the QRS complex The second group is slightly more prominent and occurs 0 04 sec later The second group of vibrations occurring 0 11 sec after the onset of the QRS probably represents an early systolic ejection sound Note the systolic murmur (SM) of low intensity and medium frequency the well split (0 04 sec) second sound (2) consisting of two groups of moderate intensity vibrations and the diastolic murmur (DM) of low intensity and medium frequency

ishes or may even disappear with expiration There are two obvious possibilities for the causation of this murmur (1) Blood flow across the atrial defect from the left to the right atrium, and (2) increased flow through the tricuspid valve, giving relative tricuspid stenosis

The timing of this murmur is such that it coincides with the period of rapid ventricular filling, that is to say, it commences with the downstroke of the wave of the right atrial pressure tracing This is not usually the time of maximal pressure difference between the left and right atria, and, therefore, is not the time of greatest flow across the defect

There are two main factors against a mitral origin for the murmur

1 Although a mid-diastolic murmur is constantly noted with large atrial shunts the presence of organic mitral stenosis is very rare

2 If the patient with atrial defect performs a Valsalva maneuver there is a decrease in venous return to the heart, with diminution, or even disappearance, of the mid diastolic rumble *If the positive intrathoracic pressure is then released* the diastolic rumble returns within two or three beats whereas mitral diastolic murmurs do not return until after four to six cardiac beats This evidence while not conclusive does suggest that the murmur has its origin in the right side of the heart

Three factors favor a tricuspid or atrial defect origin of the murmur

1 The murmur disappears after successful surgical closure of the defect with consequent reduction in flow through the tricuspid orifice and across the defect

2 The intensity of the murmur diminishes with the development of pulmonary vascular obstruction and reduction in the degree of the left to right shunt

3 Finally the location of the murmur at the lower left sternal border or between the apex and the lower left sternal border together with its increase during inspiration is consistent with a tricuspid origin of the murmur

PRESYSTOLIC MURMURS Presystolic murmurs may occur with either secundum or primum defects but are more common with the latter, perhaps because the shunts are generally larger in this group For these murmurs to be attributed to right atrial systole the vibrations should commence approximately 0 10 sec after the onset of the P wave as this is the usual time interval between the onset of electrical and mechanical systole in the right atrium It is said that a longer *interval of 0 12 to 0 13* sec between the onset of the P wave and the onset of the presystolic murmur indicates that left atrial systole is more likely the cause of the murmur Experience with patients having complete heart block does not confirm these conclusions

DIASTOLIC MURMURS *AT THE LEFT STERNAL BORDER* In many patients with uncomplicated secundum defects there is a moderate intensity, medium frequency rather early diastolic murmur heard best at the fourth or fifth left interspace This murmur is *soft* and diminuendo in quality and grade 1 to 2 in intensity It commences before the third sound It has been suggested that this murmur is due to pulmonary insufficiency secondary to dilatation of the pulmonary *artery and* the pulmonary valve ring The true cause of this murmur is not evident

PERSISTENT FORAMEN PRIMUM DEFECTS OR PARTIAL ATRIO VENTRICULAR CANAL (FIGS 160 AND 161)

Since this anomaly is simply a defect low in the atrial septum with additional deformities involving the mitral or tricuspid valve, or both, the typical sounds or murmurs described under secundum defects are present, with the additional murmurs of mitral or tricuspid insufficiency The murmur of mitral regurgitation is holosystolic in timing, blowing in character, and grade 3 to 4 in intensity It is conducted well to the left axilla and left lung base

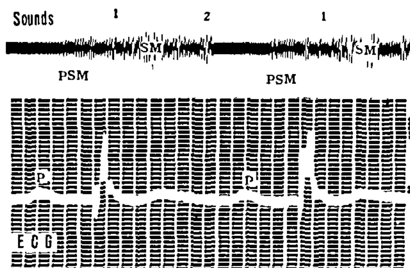


Fig 160 (Apex Log) Presystolic murmur and diamond shaped systolic murmur recorded in patient who had an ostium primum defect Note the low intensity first heart sound (1) 0 06 sec after the onset of the QRS complex the low intensity second sound (2) not split at the apex the diamond shaped systolic murmur (SM) of medium frequency and moderate intensity the medium frequency presystolic murmur (PSM) commencing 0 08 sec after the onset of the P wave of the electrocardiogram and the P R interval of 0 20 sec

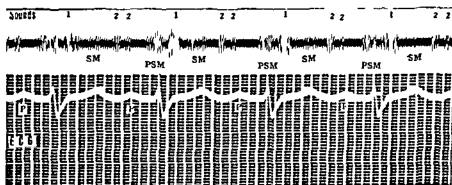


Fig 161 (Apex Log) Delayed first heart sound well split second sound and presystolic murmur in a patient with an ostium primum defect Note the first heart sound (1) of normal intensity occurring 0 09 sec after the onset of the QRS complex the systolic murmur (SM) of moderate to low intensity and medium frequency the well split (0 06 sec) second sound (2 2) of moderately low intensity with the aortic component slightly louder than the pulmonic and the moderate intensity presystolic murmur (PSM) commencing 0 12 sec after the onset of the P wave but having no presystolic crescendo

and may be accompanied by a thrill If tricuspid regurgitation is present, a holosystolic murmur may be heard at the lower left sternal border, this may be accentuated during inspiration In addition to the mid diastolic murmur heard with secundum defects, the primum defects are often accompanied by a presystolic murmur It should be stressed that patients with primum defects are more likely to have pulmonary vascular obstruction at an early age than are those with secundum defects, the Graham Steell murmur of pulmonary insufficiency also may be present

ATRIAL SEPTAL DEFECT ASSOCIATED WITH MITRAL STENOSIS (LUTEMBACHER'S SYNDROME)

The association of mitral stenosis with atrial septal defect is not common It is difficult to distinguish clinically from a secundum atrial defect However, if the diastolic rumble is unusually loud, if it has a typical presystolic crescendo with a loud apical first sound, or, if an opening snap is audible concomitant mitral stenosis may be suspected

ATRIAL SEPTAL DEFECT ASSOCIATED WITH PULMONARY VASCULAR OBSTRUCTION (FIGS 162 163, AND 164)

Pulmonary vascular obstruction causes several striking changes in the sounds and murmurs accompanying atrial septal defects These changes are due to three factors

1. Pulmonary artery hypertension
2. Diminution of the left to right shunt
3. *Pulmonic valve insufficiency*

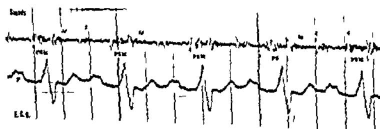


Fig 162 (Apex Log) Quadruple rhythm with widely split second sound and an atrial sound together with a normal first sound and a presystolic murmur in a patient with atrial septal defect The patient had a bidirectional shunt right ventricular hypertension $\frac{97}{(11/15)}$ right atrial a wave of 17 mm and pulmonary vascular obstruction Note the normal first sound (1) 0 09 sec after the onset of the QRS complex the unimpressive decrescendo systolic vibrations (SM) between the first and second sound the widely split (0 07 sec) second sound (2 2) the low intensity third sound (3) the prominent atrial sound (4) and the prominent presystolic murmur (PSM) commencing 0 18 sec after the onset of the P wave but having no presystolic crescendo The P R interval equals 0 22 sec, and the QRS interval 0 11 sec



Fig 163 (2 L I S Log) Early short decrescendo pulmonic diastolic murmur in a patient with atrial septal defect and pulmonary vascular obstruction Note that the first sound (1 1) consists of two sets of vibrations The first group is of low intensity and probably represents the true first sound occurring 0 08 sec after the onset of the QRS complex The second group is of high intensity occurs 0 11 sec *after the onset of the QRS and probably represents* an early systolic click Note the widely split (0 07 sec) second sound (2 2) with a markedly accentuated second component the diamond shaped systolic murmur (SM) of moderate in tensity and medium frequency and the short early decrescendo diastolic murmur (DM) com mencing with pulmonic closure and occupying only the first 20 per cent of diastole

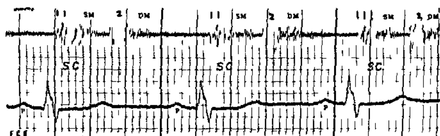


Fig 164 (2 L I S Log) Long decrescendo pulmonic diastolic murmur in patient with atrial septal defect and pulmonary artery hypertension The first sound consists of two mod erately intense series of vibrations 0 04 sec apart occurring 0 08 and 0 12 sec respectively after the onset of the QRS complex of the electrocardiogram This split first sound is followed 0 04 sec later by a high intensity group of vibrations occurring 0 16 sec after the onset of the QRS complex and representing an early systolic click (SC) Note the decrescendo sys tolic murmur (SM) of medium frequency and moderate intensity the markedly accentuated second sound (2) with slight splitting observed in some cycles and with accentuation of the second component and the long decrescendo diastolic murmur (DM) of high frequency and moderate intensity

Commensurate with the increase in pulmonary artery pressure, the force of pulmonic valve closure also increases, consequently, the second or pul monic component of the second sound at the pulmonary area may become markedly accentuated If, at the same time, pulmonary flow is greatly dimin ished, there may be some narrowing of the split That this does not happen invariably may be due to prolongation of right ventricular systole secondary *to the severe pulmonary vascular obstruction*

The diastolic murmurs associated with the left to right shunt may be greatly diminished or may disappear entirely in the presence of pulmonary vascular obstruction An early systolic click, best heard at the second left interspace or along the left sternal border, may become clearly audible This click is associated with systolic ejection into the dilated and hyper

tensive pulmonary artery and is frequently confused with splitting of the first heart sound

ATRIAL SEPTAL DEFECT ASSOCIATED WITH PARTIAL ANOMALOUS PULMONARY VENOUS DRAINAGE (FIG 165)

If the pulmonary veins from one lung (usually the right) drain into the right atrium, the findings on auscultation are identical with those of a foramen secundum atrial septal defect The diagnosis can be made only by passing a catheter into one of the veins, but the frequent association of atrial septal defect and partial anomalous pulmonary venous drainage makes a clinical diagnosis virtually impossible

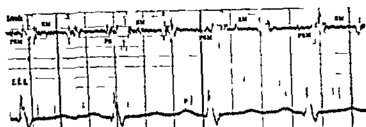


Fig 165 (2 L I S Log) Well split second sound prominent first sound and presystolic murmur in 18 year old white girl with atrial septal defect and partial anomalous pulmonary venous drainage (the pulmonary veins from the right lung entered the right atrium) Note the first sound (1 1) split by 0 05 sec (with the first component of the split much louder than the second) occurring 0 08 sec after the onset of the Q wave the systolic murmur (SM) of low intensity and low to medium frequency the well split (0 05 sec) second sound (2 2) with the two components of normal intensity and the presystolic murmur (PSM) of low frequency and low intensity having no presystolic crescendo

EFFECTS OF SURGICAL CORRECTION ON THE SOUNDS AND MURMURS OF ATRIAL SEPTAL DEFECT

If perfect surgical closure of an atrial septal defect is obtained, one would expect complete disappearance of the mid diastolic and presystolic murmurs and a reduction of the pulmonic systolic murmur to the grade 1 or 2 often found in normal persons The wide splitting of the second sound at the pulmonic area should also return to normal, especially if the delayed pulmonic closure is due entirely to increased right ventricular flow We have not followed a large enough series postoperatively entirely to confirm these opinions but certainly these results have occurred in the patients we have studied except that wide splitting of the second sound at the pulmonic area occasionally persists

REFERENCE

1 Leatham A and Gray I Auscultatory and phonocardiographic signs of atrial septal defect Brit Heart J 18 193 1956

21 / Complete Transposition of the Pulmonary Veins and of the Aorta and Pulmonary Artery

THE PULMONARY VEINS

The general clinical picture associated with complete transposition of the pulmonary veins is well known In this anomaly there is complete mixing of the systemic and pulmonary blood flow in the right atrium In order for these patients to survive there must be further communication between the two circulations, and this is almost invariably at the atrial level It is obvious that under these conditions pulmonary flow will be greater than normal, therefore, murmurs due to increased flow through the great veins and the tricuspid and pulmonic valves might be expected Audible sounds may re sult from increased right ventricular filling and from the increased force of right atrial contraction

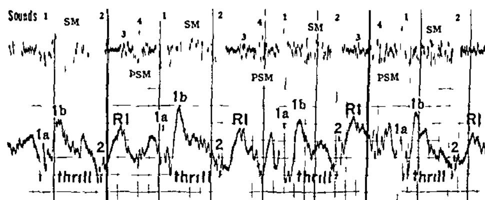


Fig 166 (2 L I S Log and apex cardiogram) Loud crescendo decrescendo systolic murmur quadruple rhythm and presystolic murmur in 2 month old girl with complete absence of the atrial septum Diagnosis confirmed at postmortem examination Note the first heart sound (1) of normal intensity the holosystolic murmur (SM) of moderate intensity and high frequency with a corresponding thrill recorded in the lower tracing the second sound (2) of normal intensity without obvious splitting the third sound (3) at the apex of the rapid inflow wave (RI) of the apex cardiogram the loud fourth sound (4) and the moderate intensity presystolic murmur (PSM)

This hemodynamic situation is similar to that resulting from a single atrium (Fig 166) The auscultatory phenomena to be described may be encountered in this situation, as well

AUSCULTATION (FIGS 167 TO 170)

SOUNDS The first sound is normal in intensity and is loudest at the lower left sternal border or at the apex The second sound is usually prominent, with normal splitting In the presence of pulmonary vascular obstruction pulmonic closure is accentuated A clear third sound (presumably secondary to increased right ventricular filling) is commonly encountered at the lower *left sternal* border or the apex *Fourth* sounds are heard in at least half of the patients giving a quadruple rhythm but in infants and children the tachycardia often encountered gives rise to a summation gallop—a triple rather than a quadruple rhythm

MURMURS Three types of murmurs are frequently encountered (1) a holosystolic *murmur*, diamond shaped or decrescendo loudest over the pulmonic area and presumably due to increased flow through the pulmonic

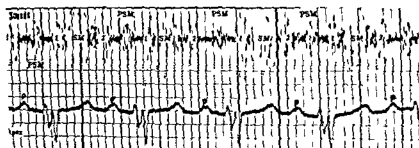


Fig 167 (L L S B Log) Characteristic diamond shaped systolic murmur and diastolic presystolic murmur in patient with anomalous pulmonary venous drainage Note the loud first sound (1) 0 08 sec after the onset of the Q wave the diamond shaped high intensity medium to high frequency systolic murmur (SM) the prominent second sound (2) split by 0 03 sec and the late diastolic presystolic murmur (PSM) of medium to high frequency and moderate intensity probably accentuated by the short diastole with the P wave occurring early in diastole and giving a summation effect

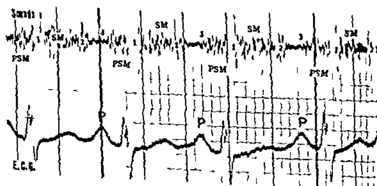
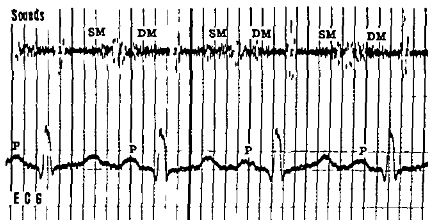
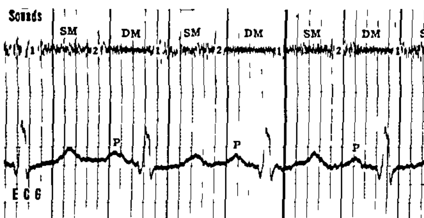


Fig 168 (L L S B Log) *Decrescendo* systolic and presystolic murmur in patient with total anomalous pulmonary venous drainage Note the prominent first sound (1) 0 08 sec after the onset of the QRS complex *the normal second sound* (2) *the low intensity third* sound (3) *the systolic murmur* (SM) *of medium* frequency and moderate intensity and the presystolic murmur (PSM) also of *medium* frequency and moderate intensity

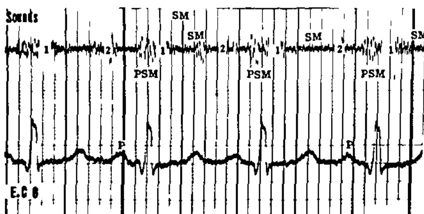
Baby C 4 Months



Under Left Clavicle



3rd Left Interspace



Apex

Fig 169 (Logarithmic tracing) A continuous murmur under the left clavicle and the more typical rhythm at the apex in a patient with total anomalous pulmonary venous drainage Note the normal first sound (1) and the continuous murmur under the left clavicle with accentuation of the murmur at the time of the second sound the normal first (1) and second (2) sounds and a less intense continuous murmur at the third left interspace and the normal first (1) and second (2) sounds with a somewhat diamond shaped systolic murmur (SM) and a presystolic murmur (PSM) at the apex

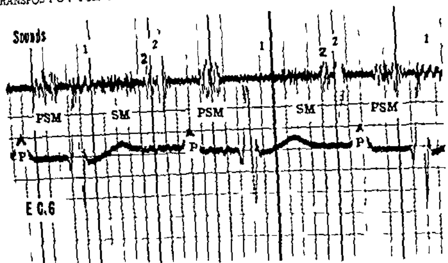


Fig 170 (L.L S B Log) First degree heart block in total anomalous pulmonary venous drainage The P R interval was 0 20 sec at a ventricular rate of approximately 110 beats per minute Note the low intensity first sound (1) the crescendo decrescendo systolic murmur (SM) of low intensity and medium frequency the well split (0 04 sec) second sound (2 ") with an accentuated second component and the presystolic murmur (PSM) falling late in diastole rather than in true presystole This murmur commences about 0 08 sec after the onset of the P wave and ends about 0 03 sec before the onset of the first heart sound It is crescendo decrescendo in configuration

valve, (2) a mid diastolic presystolic murmur over the tricuspid area, probably due to increased flow through the tricuspid valve, and (3) a continuous murmur over the great veins and under the right or left clavicle or both, which is actually a venous hum in the large venous channels

It should be emphasized that these characteristic sounds and murmurs have been encountered in those cases of complete transposition of the pulmonary veins when drainage is into the right or left superior vena cava, into the coronary sinus or directly into the right atrium We have not heard these murmurs where drainage has been below the diaphragm, into the portal vein or the ductus venosus

THE AORTA AND PULMONARY ARTERY

The diagnosis of complete transposition of the aorta and pulmonary artery is made from the over all clinical picture, but auscultation may be helpful in pointing to associated anomalies For patients with complete transposition to survive there must be communications between the two circulations, thus ventricular defects patent ductus arteriosus, or atrial defects, alone or in combination, are frequently encountered

Auscultation

The first sound is normal in intensity The second sound is usually split and may be normal, diminished, or accentuated in intensity Because of the

anomalous position of vessels and the variable hemodynamics, it is not possible to assume that the sounds at the second left and second right interspaces reflect pulmonic and aortic valve closure To complicate further this problem, we are not even sure whether aortic or pulmonic valve closure occurs first in these patients

Although the heart sounds are not particularly helpful in diagnosis, the murmurs, which occur in about 70 per cent of such patients, may be more so In general these murmurs reflect the associated anomalies, and, since ventricular defects are common, so are systolic murmurs at the lower left sternal border Diastolic murmurs are rare but may occur in individuals with large left to right shunts at the atrial or ventricular level If there is stenosis of either the aortic or pulmonary outflow tracts, an associated stenotic murmur will be present

22 / Coarctation of the Aorta

Although coarctation of the aorta (Fig 171) is never diagnosed on the basis of auscultation alone, careful auscultation of the accompanying heart sounds and murmurs enables one to assess better the nature of lesions which are commonly associated with coarctation

AUSCULTATION

First heart sound

The first heart sound may be accentuated at the apex because of the hyperactive beat necessary to maintain the associated hypertension in the proximal aorta

Second heart sound

Aortic valve closure is usually accentuated because of the elevated diastolic pressure in the proximal aorta whereas pulmonic closure is normal in intensity unless there is associated pulmonary artery hypertension secondary to congestive failure mitral valve disease, pulmonary vascular obstruction or a left to right shunt

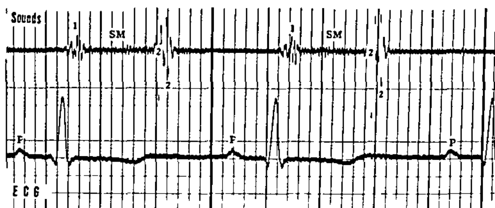
Murmurs

Systolic A late systolic murmur, grade 2 to 3 in intensity may be heard in the fourth or fifth left interspace or even at the apex It is possible that this murmur represents flow across the coarcted segment itself or even more likely associated mitral regurgitation

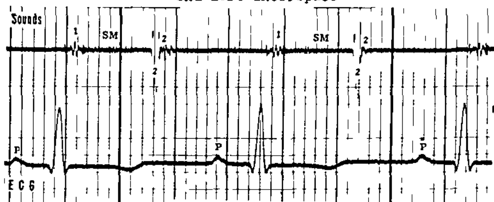
A stenotic systolic murmur grade 2 to 3 in intensity, is heard frequently in the aortic area it is conducted well into the suprasternal notch along the great vessels of the neck and into the back This murmur may be due to co existing aortic stenosis or it may form in the dilated ascending aorta Brock[1] described a patient with coarctation of the aorta who had a prominent stenotic aortic systolic murmur accompanied by a thrill, but no gradient across the aortic valve

Diastolic Aortic diastolic murmurs are common and are usually due to the association of aortic hypertension and a bicuspid aortic valve which also may be the site of bacterial endocarditis Apical diastolic murmurs are noted in approximately 25 per cent of patients who have coarctation of the

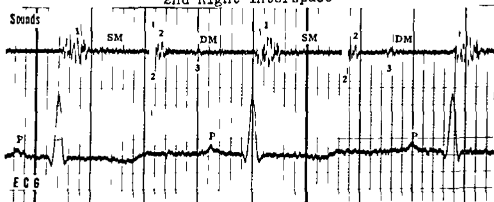
R.A 13 Yrs



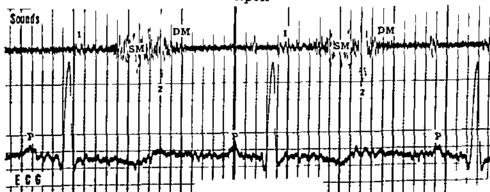
2nd Left Interspace



2nd Right Interspace



Apex



Left Upper Back

aorta These murmurs are mid diastolic, low frequency, moderate intensity rumbles and may be due to left ventricular dilatation or to associated mitral valve disease

CONTINUOUS MURMURS Continuous murmurs are frequently associated with coarctation, and depending on the position of maximal intensity, they may be due to one of three possible causes

1 A continuous murmur along the left or the right sternal border may be due to collateral flow through the internal mammary arteries and similar murmurs over the intercostal spaces especially in the paravertebral region may be due to flow through intercostal arteries

2 A continuous murmur in the left upper part of the chest anteriorly or over the left upper part of the back could be secondary to flow across the severely coarcted segment If both the systolic and diastolic pressures proximal to the coarctation were greater than those in the distal segment of the aorta a gradient would persist across the coarctation throughout the cardiac cycle and a continuous murmur could result

3 A continuous murmur in the second left interspace or in the left infraclavicular region may be due to an associated patent ductus arteriosus

REFERENCE

1 Brock Russell *Surgical treatment of aortic stenosis* Brit M J 1 1019 1957

Fig 171 Phonocardiograms of patient with coarctation of the aorta

At the second left interspace the first sound (1) is of normal intensity and the second sound (2) is split by 0 04 sec with the aortic or first component louder than the second or pulmonic component There is rather low intensity medium frequency systolic murmur (SM) lasting throughout all of systole

At the second right interspace the splitting of the second sound (2) is less obvious as the first or aortic component is now intense whereas pulmonic closure is barely recorded

At the apex the loud aortic component of the second sound (2) is still dominant but there is now recorded a low intensity *third sound* (3) and a low intensity mid diastolic murmur (DM)

Over the left upper portion of the back there is the continuous murmur (SM DM) with its late systolic accentuation This murmur could not be obliterated by pressure on the intercostal arteries close to its point of maximal intensity and it is possible that it was generated at the site of the coarctation

23 / Congenital Aortic Stenosis

Congenital aortic stenosis may be valvular, subvalvular, or supravalvular, it is not possible to differentiate these types clinically We have studied more than 150 patients,[1] basing our diagnoses on the following criteria

1 A 'stenotic systolic murmur heard at the second right interspace with wide transmission
2 The electrocardiogram shows left ventricular dominance or left ventricular hypertrophy
3 No evidence of a left to right or right to left shunt

AUSCULTATION

First heart sound

Frequently the first sound is loud at the apex and appears widely split Simultaneous phonocardiograms, electrocardiograms, and arterial pressure tracings suggest that, whereas the first part of the split first sound seems to coincide with mitral valve closure, the second part coincides with systolic ejection, and so must occur after aortic valve opening The latter sound is referred to as "an early systolic ejection sound" or "systolic click" and

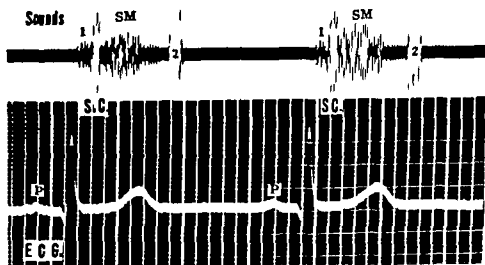


Fig 172 (2 R I S Log) The early systolic click of congenital aortic stenosis Note the first sound (1) of low intensity 0 06 sec after the onset of the QRS complex the loud early systolic click (SC) 0 10 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) of medium frequency and moderate intensity ending before the second sound and the second sound (2) of normal intensity split by 0 02 sec

probably arises in the aorta itself (Fig 172) It corresponds to the early systolic ejection sound heard over the pulmonic area in patients who have dilatation of the pulmonary artery, and it occurs about 0 05 sec *after mitral* closure

AORTIC SECOND SOUND

In two thirds of the patients, the aortic second sound was less intense than the pulmonic second sound, but it was considered to be definitely diminished in only one third (Fig 173) It was usually narrowly split, but often appeared single The timing of aortic valve closure more closely approximates that of pulmonic valve closure than it does normally, and because of the prolonged left ventricular systole, it may occur even after pulmonic valve closure, giving rise to paradoxical splitting with inspiration

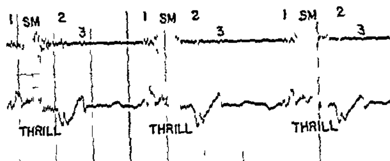


Fig 173 (2 R I S Log and In er) The systolic thrill of congenital aortic stenosis Note the low intensity *first sound (1)* the diamond shaped systolic murmur (SM) with the apex of the diamond in the first third of systole the low intensity second sound (2) and the third sound (3) A systolic thrill is clearly shown on the lower tracing

SYSTOLIC MURMUR

The *systolic murmur* of congenital aortic stenosis is grade 3 or louder and has a harsh rough quality (Fig 174) This murmur is heard best at the second right interspace and is conducted well to the *suprasternal* notch and along the great vessels of the neck, it is of medium frequency reaches its point of *maximal intensity* before midsystole and tails off before the second heart sound In several proved cases the murmur has been loudest at the mid left sternal border

By *phonocardiography*, it can be seen that the *murmur* begins immediately after the first sound and is diamond shaped with the apex of the diamond occurring before midsystole We have not been able to confirm the impression of the Swedish authors that the apex of the diamond occurs later in systole in the more severe cases than it does in the milder cases Lesser degrees of aortic *stenosis may* be accompanied by less intense murmurs without thrills but because *of the criteria we used for diagnosis*, most of our patients had a rough systolic thrill *felt* best at the second right interspace accompanying the murmur (Fig 174)

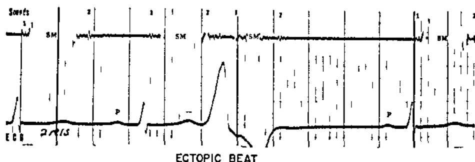


Fig 174 (2 R I S Log) Effect of a ventricular premature beat on the systolic murmur of congenital aortic stenosis Note the low intensity first sound (1) 0 05 sec after the onset of the QRS complex and the low intensity second sound (2) In the normally conducted beats there is a diamond shaped systolic murmur (SM) of high intensity and high frequency with the apex of the diamond around mid systole With the ectopic beat falling soon after the previous complex the first sound occurs 0 19 sec after the onset of the QRS complex and the systolic murmur is considerably diminished in intensity Mechanical systole is shortened whereas electrical systole is prolonged secondary to the marked conduction defect

Diastolic murmurs

Decrescendo aortic diastolic murmurs (Fig 175) occurred in 15 to 20 per cent of our patients Postmortem examination of some of the valves showed that in certain cases the valve leaflets could not close completely because of

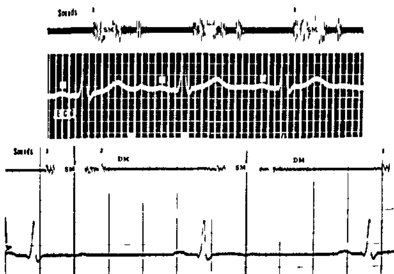


Fig 175 Preoperative and postoperative phonocardiograms of a patient who had congenital aortic stenosis showing the development of a low intensity aortic diastolic murmur following transventricular splitting of the aortic valve

Upper tracing (2 R I S Log) The first high frequency vibrations (1) occur 0 12 sec after the onset of the Q wave and probably represent an early systolic click Note the diamond shaped systolic murmur (SM) of high frequency and moderate intensity with the apex of the diamond in the first third of systole and the normal intensity second sound (2)

Lower tracing (2 R I S Log) The initial high frequency vibrations (1) again occur 0 12 sec after the onset of the Q wave The diamond shaped systolic murmur (SM) is more intense than it was preoperatively and it appears to start later in relation to the Q wave The second sound (2) is essentially unchanged A diastolic murmur (DM) of low intensity and medium frequency starts with the second sound and lasts through half of diastole

structural deformity therefore, the presence of aortic insufficiency was not surprising It is important to remember that the murmur of aortic incompetence is heard at least as frequently, if not more commonly, with subvalvular as with valvular stenosis

Apical diastolic murmurs are relatively rare and are probably due to left ventricular dilatation, or perhaps to involvement of the mitral valve in some process such as endocardial fibro elastosis

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24 / Congenital Mitral Stenosis

Congenital mitral stenosis is rarely encountered as an isolated lesion Fre quently it is associated with *endocardial fibro elastosis*

AUSCULTATION

Sounds

In the presence of a pliable mitral valve the first sound may be accentu ated (Fig 176), but in 1 of the 2 patients (Fig 177) with a flat mem branous type of mitral valve, immobile to the surgeon's palpating finger, on whom we have operated, the first sound was diminished in intensity The second sound at the pulmonic area is moderately to markedly accentuated, *depending on the degree of pulmonary artery hypertension The third sound,* if present, is of low intensity An opening snap may or may not be present, depending on the mobility of the mitral valve

Murmurs

The apical diastolic murmurs in our patients with pure *stenosis have been* loud, sometimes with presystolic accentuation, however, patients with asso ciated endocardial fibro elastosis, who have been in failure, have had low intensity murmurs

DIFFERENTIATION BETWEEN CONGENITAL AND RHEUMATIC MITRAL STENOSIS

The essential factor in the differential diagnosis is the age at which the lesion is detected, classical murmurs of pure mitral stenosis are much more likely to be congenital in origin if they occur in children less than five years of age than if they occur in older children

Cor triatriatum

We have encountered 2 patients in whom the left atrium was divided by a membrane passing from the interatrial septum to the lateral wall of the left atrium and obstructing blood flow from the pulmonary veins to the mitral valve In each instance there was a small hole in the membrane Both patients had *clinical pictures identical with that of primary pulmonary*

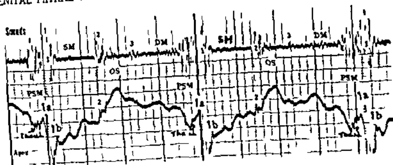


Fig 176 (Apex Log and apex cardiogram) The typical murmurs associated with severe congenital mitral stenosis Note the extremely loud first sound (1) a few systolic vibrations (SM) the moderately loud second sound (2) split by 0 04 sec the low intensity opening snap (OS) (this does not coincide with any obvious valley or O point in the apex cardiogram) the low intensity third sound (3) a diastolic murmur (DM) of low frequency and low intensity during middiastole and a presystolic murmur (PSM) of high intensity with a thrill recorded simultaneously on the apex cardiogram as a series of vibrations preceding the 1a point



Fig 177 (Apex Log) Intense middiastolic presystolic murmur 6 year old boy with congenital mitral stenosis Note the rather low intensity first sound (1) 0 08 sec after the Q wave the normal second sound (2) split by 0 03 sec and the diastolic murmur (DM) of extremely high intensity and medium frequency starting about the expected time of the opening snap and continuing to the succeeding first sound but without presystolic crescendo

vascular obstruction and both died within the first two months of life In each case the only abnormal auscultatory finding was an accentuation of pulmonary valve closure

Congenital mitral stenosis was considered in the differential diagnosis of these patients but was rejected because of the absence of murmurs and because the left atrium was normal in size An identical picture occurs with stenosis of the pulmonary veins prior to their entry into the left atrium

We have seen 1 additional patient in whom left atrial stenosis resulting from a thickened ring of fibro elastic tissue was just proximal to the mitral valve The clinical signs and murmurs were identical with those of mitral stenosis except that there was no record of an opening snap on auscultation A phonocardiogram was not obtained

Congenital mitral insufficiency

Pure mitral insufficiency is a rare congenital anomaly and is usually secondary to congenital shortening of the chordae tendineae, or to maldevelopment of the mitral cusps

Mitral insufficiency, often of severe degree, may accompany the secundum type of atrial septal defect or the usual form of ventricular defect Cleft mitral valves are much more commonly encountered as part of the syndrome of common atrial ventricular canal, of the partial or complete variety As part of the syndrome of corrected transposition a left sided Ebstein's deformity may occur giving left A V valve insufficiency

Any cause of left ventricular failure with dilatation of the mitral ring, the presence of endocardial fibro elastosis with mitral valve involvement, or destruction of the mitral valve secondary to bacterial endocarditis, may lead to mitral insufficiency of varying degree

25 / Pulmonic Stenosis and Tetralogy of Fallot

It is important to differentiate clinically between pulmonic stenosis with intact ventricular septum and pulmonic stenosis with a ventricular septal defect, since the surgical approach to these lesions may be different For similar reasons, it is important to differentiate valvular from infundibular pulmonic stenosis. It should be stated that, although many points of difference will be described, there are times when it may be impossible to make the differential diagnosis with certainty

In this chapter we use the term "pure pulmonic stenosis" for pulmonic stenosis with an intact ventricular septum even though there may be a patent foramen ovale The term "tetralogy of Fallot" refers to pulmonic stenosis associated with a ventricular septal defect and a right to left shunt We will not discuss, in this chapter those cases where there is a left to right shunt through a ventricular defect, together with a gradient across the pulmonic valve

AUSCULTATION

First heart sound

In pulmonic stenosis with intact ventricular septum (Fig 178) In pure pulmonic stenosis, either valvular or infundibular, the first sound is often loudest at the lower left sternal border, where it may be normal or accentuated

Increased intensity of the first sound in this area may be due to the increased force of right ventricular contraction causing a sharp closure of the tricuspid valve this is comparable to the increased intensity of the mitral first sound in hyperdynamic hearts Another possible factor which may contribute occasionally to the loud first sound in valvular stenosis is the semilunar component of the first sound caused by the opening of the pulmonic valves In some patients who have pulmonic valve stenosis the valve is pliable having a slight convexity toward the right ventricle during diastole and ballooning toward the pulmonary artery in systole however, it cannot open fully because of the basic structural deformity These changes in the position of the valve have been demonstrated nicely by Kjellberg and his associates[1] using angiocardiography

Early systolic click or protosystolic clap In mild valvular pulmonic stenosis with intact ventricular septum and post stenotic dilatation of the

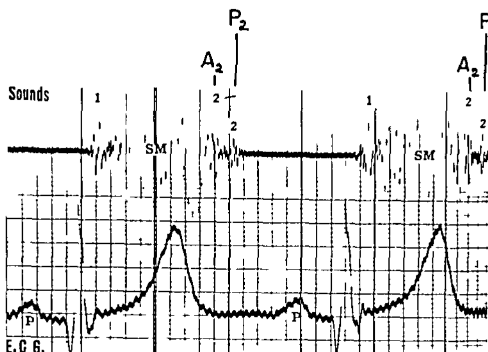


Fig 178 (2 L I S Log) Typical diamond shaped systolic murmur with a well split second sound in a patient with pure valvular pulmonic stenosis Note the normal first sound (1) 0 08 sec after the Q wave of the electrocardiogram the well split second sound (° 2) split by 0 05 sec the aortic closure (A_2) louder than pulmonic closure (P_2) and the diamond shaped systolic murmur (SM) of high frequency and high intensity commencing a definite interval after the first sound The apex of the diamond is in mid systole

pulmonary artery, the first sound may appear accentuated over the region of the pulmonary artery This loud sound, which was described by Petit in 1902, immediately follows the true first heart sound [3]

The combination of the post stenotic dilatation and the jet effect of the blood suddenly entering the pulmonary artery results in the production of an early systolic click This click, called the ejection sound, since it coincides with filling of the pulmonary artery is often palpable as a shock or thrust over the region of the pulmonary artery Probably the presence or absence of this click depends on the interplay of several factors, such as (1) the angle at which the jet of blood is directed from the pulmonic valve into the pulmonary artery, (2) the degree of post stenotic dilatation of the pulmonary artery, and (3) the force of right ventricular contraction The click becomes diminished during inspiration and may disappear entirely during a Valsalva maneuver although it persists during normal expiration

In tetralogy of Fallot The first sound is usually of normal intensity in tetralogy of Fallot, although it may be accentuated at the lower left sternal border Splitting is either narrow or not present Following the first sound there may be an early systolic ejection sound which is well conducted along the carotids, this sound probably occurs in the aorta itself An important factor in the causation of this sound may be the increased stroke

output into the somewhat dilated aorta, and its clarity may be increased by the close proximity of the aorta to the anterior wall of the chest

PULMONIC SECOND SOUND

IN PULMONIC STENOSIS WITH INTACT VENTRICULAR SEPTUM (FIG 179) In mild pulmonic stenosis the second sound at the pulmonic area is moderately well split, and pulmonic closure may be either normal or diminished

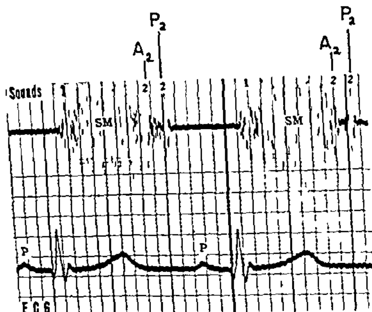


Fig 179 (2 L.I.S. Lg.) Typical diamond shaped systolic murmur with a well split second sound in a patient with pure valvular pulmonic stenosis Note the normal intensity first sound (1) with the main valvular component occurring 0 08 sec after the onset of the Q wave and the systolic murmur (SM) of high frequency and high intensity continuing through to the second valvular component of the *second sound* The murmur starts with a prominent component 0 04 sec after the main valvular component of the first sound It is diamond shaped with the apex of the diamond occurring near mid systole Note also the widely split second sound (2 2) split by 0 06 sec the aortic valve closure (A) and the pulmonary valve closure (P)

in intensity the splitting usually increases with inspiration When the ventricular septum is intact valvular or infundibular pulmonic stenosis of severe degree may result in a greatly diminished, inaudible, or even unregisterable pulmonic valve closure because of two main factors (1) low pulmonary artery pressure, and (2) structural abnormality of the valve itself

In severe pulmonic stenosis, prolonged right ventricular systole causes wide splitting of the second sound in the pulmonic area This splitting is due to delayed pulmonic valve closure and does not increase appreciably on inspiration

IN TETRALOGY OF FALLOT Aortic and pulmonic valve closures occur al

most simultaneously in tetralogy of Fallot Aortic valve closure occurs first, since aortic pressure is much higher than pulmonary artery pressure, but because of the ventricular septal defect, right and left ventricular pressures fall rapidly together, and the interval between aortic and pulmonic valve closure is extremely short Also, since pulmonic valve closure occurs at a much lower pressure than aortic valve closure, and since the aortic valve is anterior, the second sound is dominated by the aortic component Consequently, under these circumstances, the second sound is diminished at the pulmonic area and is best heard as a loud, narrow, or single sound at the lower left sternal border

The timing of aortic valve closure can be measured from the dicrotic notch on the carotid pulse tracing with a correction for the delayed pulse transmission, and occasionally pulmonic valve closure can be calculated from simultaneous right ventricular and pulmonary artery pressure tracings, or, better still, from selective angiocardiography with a rapid film changer, or by x ray cinematography in conjunction with selective angiocardiography

Following shunt procedures The Blalock or Potts shunt operations for tetralogy of Fallot result in increased pressure in the pulmonary artery with consequent increased intensity of pulmonic closure When the shunt is too large, there may be a considerable increase in pulmonary artery pressure, and pulmonic closure may become markedly accentuated

SYSTOLIC MURMUR OF PULMONIC STENOSIS

IN PULMONIC STENOSIS WITH INTACT VENTRICULAR SEPTUM (FIG 180) *Site of maximal intensity* In valvular pulmonic stenosis, the systolic mur

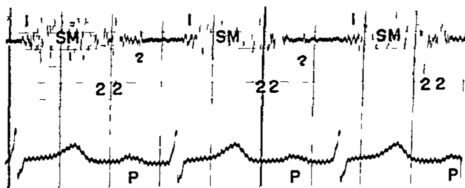


Fig 180 (2 L I S Log) Diamond shaped systolic murmur and a protodiastolic high frequency sound in a patient with pulmonic stenosis with intact ventricular septum Note the first sound (I) of low frequency and low intensity 0 06 sec after the onset of the QRS complex the diamond shaped systolic murmur (SM) of high intensity and medium to high frequency with the apex of the diamond near mid systole and the well split (0 05 sec) second sound (2 2) with some vibrations continuing through to the second component There is a narrow high frequency vibration marked with a ? which occurs 0 08 to 0 09 sec after the termination of the second sound This may be a third sound but it is of unusual frequency and occurs early Also it could be an opening snap but the reason for its presence is not obvious

mur is heard best at the first and second left interspaces and is conducted well to the neck and back It is almost invariably accompanied by a thrill

Character of the murmur The murmur is loud, usually grade 3 or 4 in intensity, and has a rough, stenotic quality It is of medium to high frequency on the phonocardiogram, begins some time beyond the first sound, and is diamond shaped usually with the apex of the diamond at or beyond the midpoint of systole if the stenosis is severe It is louder than the murmur found in similar degrees of pulmonic stenosis, associated with ventricular septal defect This is probably because there is less blood flowing through the pulmonic valve orifice in tetralogy of Fallot It must be emphasized that the loudness of the murmur is not an index of the severity of the stenosis in fact, the most severe lesions, especially when accompanied by congestive failure, may have unimpressive murmurs

Duration of the murmur The systolic murmur of pulmonic stenosis must end at or before the time of pulmonic valve closure, in patients with severe pulmonic stenosis, the murmur, continuing throughout the whole of right ventricular systole, may obscure aortic valve closure

IN TETRALOGY OF FALLOT Pulmonic stenosis with a ventricular septal defect is usually infundibular, but may be valvular or a combination of the two

Site of maximal intensity In infundibular stenosis the systolic murmur is heard best at the third and fourth interspaces at the left sternal border, frequently it resembles closely the murmur of ventricular septal defect At times it may be difficult to decide whether stenosis is valvular, infundibular, or both

Character of the murmur The murmur is stenotic in character and diamond shaped on the phonocardiogram The apex of the diamond is usually early and the murmur itself begins immediately with the first sound Usually it is not as loud as the murmur of pulmonic stenosis with intact ventricular septum the intensity is rarely more than grade 3 and it varies inversely with the severity of the lesion Patients who have severe tetralogy of Fallot may have murmurs of only grade 1 to 2 in intensity, and, of course, in the most severe form pulmonary atresia with ventricular septal defect, there may be no murmur at all Patients with severe tetralogy who have spells of cyanosis and perhaps unconsciousness, may lose their systolic murmur during the attacks

Duration of the murmur The systolic murmur of tetralogy of Fallot does not continue beyond aortic valve closure, which is virtually simultaneous with pulmonic closure

Conduction of the murmur It has been stated [4] that the wideness of transmission of a murmur is due solely to its loudness and that the direction of blood flow is not important in this regard A form of tetralogy of Fallot with absence of the left main pulmonary artery has been instructive in this respect Patients with this condition show transmission of the murmur to the

right upper part of the back and to the right infraclavicular region far better than to the left upper part of the back or the left infraclavicular region It is believed, therefore, that, although loudness of a murmur is the dominant factor in transmission, nevertheless, direction of blood flow is a significant contributing factor

Presystolic sounds and murmurs in pulmonic stenosis with intact ventricular septum (Fig 181)

In severe cases of pure pulmonic stenosis associated with right ventricular failure, right atrial hypertension, and possibly a right to left shunt through a patent foramen ovale, one occasionally encounters an atrial sound (presystolic gallop) or an actual presystolic murmur Two possible explanations are suggested for the origin of the murmur It may be due (1) to the right to left shunt through the patent foramen ovale at the time of atrial systole, or (2) to flow through a small tricuspid valve, or even through the pulmonic valve, secondary to forceful atrial systole

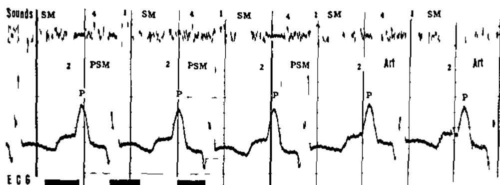


Fig 181 (2 L I S Log) Systolic and presystolic murmurs together with a presystolic gallop in an infant with pure pulmonic stenosis congestive failure and a right to left shunt through a patent foramen ovale Right ventricular pressure was 180 mm of mercury Note the normal first sound (1) 0 07 sec after the onset of the QRS complex the holosystolic diamond shaped systolic murmur (SM) of medium to high frequency and moderate intensity the normal intensity apparently single second sound (2) the loud atrial sound (4) following by 0 08 sec the onset of the tremendous P waves of the electrocardiogram and the presystolic murmur (PSM) following the fourth sound Art represents respiratory artefact



DIFFERENTIAL DIAGNOSIS

Although it is relatively easy to be confident of one's diagnosis in severe valvular pulmonic stenosis with an intact ventricular septum, and also in many cases of clearcut tetralogy of Fallot nevertheless, there is a small group of cases where the diagnosis can be confirmed only at catheterization or by selective angiocardiography

Differentiation of mild infundibular pulmonic stenosis from a small ventricular septal defect or mild subaortic stenosis may be completely impos

sible on clinical grounds All may have a coarse, rough systolic murmur along the left sternal border, and roentgenograms and electrocardiograms may be within normal limits in each case Since it is not justifiable to catheterize these patients who have only mild symptoms, or none at all, it is likely that the diagnosis based on clinical findings may be wrong

PULMONIC INSUFFICIENCY MURMUR FOLLOWING PULMONARY VALVOTOMY

A pulmonic insufficiency blow may be heard at the pulmonic area following the operation of pulmonary valvotomy The most surprising feature is that this murmur is not encountered more frequently Even when present, it is not striking, and careful auscultation may be necessary for its detection Its appearance is often delayed after valvotomy, as it is related to the development of critical pulmonary artery pressure A pulmonary diastolic murmur invariably accompanies complete repair of tetralogy of Fallot when a prosthesis is inserted from the outflow tract of the right ventricle, across the pulmonary valve ring and along the trunk of the main pulmonary artery

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26 / Idiopathic Pulmonary Insufficiency

During the past five years we have seen 6 patients whom we believe hav idiopathic pulmonary insufficiency (*for examples see figures 182 and 183* Since all are still alive and are completely asymptomatic we do not kno the underlying pathology All patients have prominent main pulmonary a tery segments, and catheterization has revealed no abnormalities Dilatatio of the pulmonary valve ring or defective pulmonic valve cusps are possib factors in the causation of the murmur

AUSCULTATION

The heart sounds are normal and a pulmonic systolic murmur of slight, o rarely of moderate, intensity may be present The striking feature of th examination is a loud grade 3 or 4 medium to high frequency diastolic mur mur at the pulmonic area, commencing with a normal second sound an lasting throughout the greater part of diastole The configuration of th murmur on the phonocardiogram is variable but has a tendency to be de crescendo

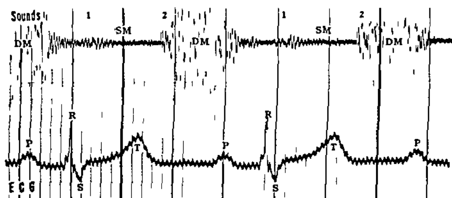


Fig 182 (2 L I S Log) Loud crescendo decrescendo diastolic murmur commencing with *the second valvular component of the second heart sound in idiopathic pulmonary valve in*sufficiency Note the low intensity first heart sound (1) 0 08 sec after the onset of the QRS complex the systolic murmur (SM) of low frequency and very low intensity the second sound (2) of moderate intensity split by 0 03 sec and the almost diamond shaped diastolic murmur (DM) of medium to high frequency and very high intensity starting with pulmonic closure and continuing throughout the first three quarters of diastole

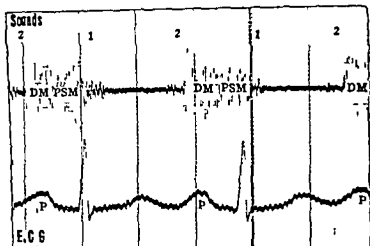


Fig 183 (2 L.I.S. Log.) Loud diastolic murmur with presystolic accentuation in idiopathic pulmonary insufficiency. Note the low intensity first heart sound (1), the very low *intensity* second sound (2) and the diastolic murmur (DM) of medium frequency and moderate intensity continuing into presystole (PSM).

26 / Idiopathic Pulmonary Insufficiency

During the past five years we have seen 6 patients whom we believe have idiopathic pulmonary insufficiency (for examples see figures 182 and 183) Since all are still alive and are completely asymptomatic we do not know the underlying pathology All patients have prominent main pulmonary artery segments, and catheterization has revealed no abnormalities Dilatation of the pulmonary valve ring or defective pulmonic valve cusps are possible factors in the causation of the murmur

AUSCULTATION

The heart sounds are normal and a pulmonic systolic murmur of slight, or rarely of moderate, intensity may be present The striking feature of the examination is a loud grade 3 or 4 medium to high frequency diastolic murmur at the pulmonic area, commencing with a normal second sound and lasting throughout the greater part of diastole The configuration of the murmur on the phonocardiogram is variable but has a tendency to be decrescendo

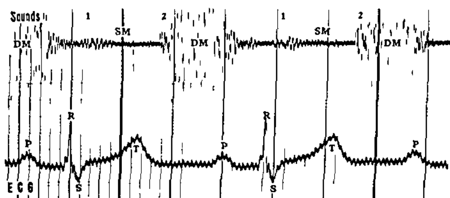


Fig 182 (2 L I S Log) Loud crescendo decrescendo diastolic murmur commencing with the second valvular component of the second heart sound in idiopathic pulmonary valve insufficiency Note the low intensity first heart sound (1) 0 08 sec after the onset of the QRS complex the systolic murmur (SM) of low frequency and very low intensity the second sound (2) of moderate intensity split by 0 03 sec and the almost diamond shaped diastolic murmur (DM) of medium to high frequency and very high intensity starting with pulmonic closure and continuing throughout the first three quarters of diastole

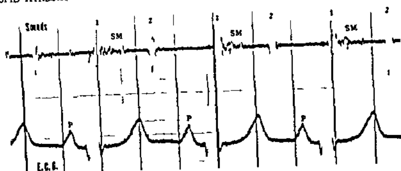


Fig 184 (4 L I S Log) The insignificant systol c murmu encountered in about half of our p tients w th t cusp d at es a Note the narrow first sound (1) of normal intens ty 0 07 sec after the onset of th QRS complex the decrescendo systolic murm r (SM) of low intens ty and m d um f equ cy occupy ng the first half of systole and the s ngle second sound (2)

INCREASED PULMONARY BLOOD FLOW

Patients who have tricuspid atresia and increased pulmonary blood flow usually have a good pulmonary outflow tract and a hypoplastic aortic out flow tract Ventricular septal defects or transposition of the great vessels are the most commonly associated lesions

Auscultation

Sounds The first sound is single, of normal intensity, and loudest at the apex The second sound at the second left interspace is usually single, but it may be of normal intensity or even accentuated Occasionally, it is heard best at the lower left sternal border

Murmurs As with the previous group, the murmurs are usually at the lower left sternal border are systolic in timing and do not appear significant in at least half of the patients

Mid diastolic and Presystolic Murmurs (Fig 185) Mid diastolic and presystolic murmurs are exceedingly rare with tricuspid atresia, but we have

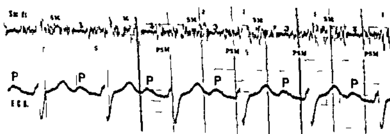


Fig 185 (Ap Log) A p esyst l c m mur n a pat ent w th tr cusp d atres a Note the n mal f st so d (1) 0 08 s c afte the ons t of the QRS compl x the systol c m mu (SM) of m d um i t s ty and mode ately h gh fr que cy the normal ntens ty second sou d (2) pl t by 0 02 sec the th d s u d (3) of low f eq ency a d low intensity a d the p esyst l c m mu (PSM) of mode ate nte s ty d m d um frequ cy commenc ng 0 12 sec afte th o set of th P wa e

27 / Tricuspid Atresia

Tricuspid atresia should be suspected in all cyanotic children in whom the electrocardiogram shows left axis deviation and left ventricular dominance or hypertrophy A thorough knowledge of the various lesions commonly associated with tricuspid *atresia and the accompanying murmurs makes it* possible to predict better the anatomic arrangement in any given patient

Tricuspid atresia itself produces no particular alterations in the heart murmurs or sounds except, of course, that there is no contribution to the first sound from tricuspid valve closure All departures from normal in auscultation are due to associated lesions, but, even in spite of these, it must be stressed that many patients with tricuspid atresia do not have significant murmurs, *in fact, in our series of over 50 patients with* proved tricuspid atresia, almost half had only grade 1 or 2 left sternal border systolic murmurs

For our purposes, tricuspid atresia is best discussed under two main headings (1) with diminished pulmonary blood flow, and (2) with increased pulmonary blood flow

DIMINISHED PULMONARY BLOOD FLOW

Patients who have tricuspid atresia and diminished pulmonary blood flow almost always have an associated ventricular septal defect, a good aortic outflow tract, and a hypoplastic pulmonary outflow tract

Auscultation (Fig 184)

Sounds The first heart sound is single, of normal intensity, and *loudest* at the apex The second sound is single is usually diminished at the upper left sternal border, and is loudest at the lower left sternal border Atrial sounds may be heard in those patients with a small patent foramen ovale and increased right atrial pressure

Murmurs Systolic murmurs are encountered in almost all patients In 50 per cent, the murmur is only grade 1 or 2 and is not remarkable, but in the remainder it *may be grade 3 or 4 and is holosystolic Presumably* this murmur is due to flow across the ventricular defect Stenotic pulmonic systolic murmurs associated with the hypoplastic pulmonary outflow tract are extremely rare

28 / Ebstein's Anomaly

During the past six years we have examined clinically 16 patients with Ebstein's anomaly and have obtained phonocardiograms on 10 of the 16. The auscultatory and phonocardiographic signs, while not diagnostic, are fairly consistent and certainly suggest the diagnosis (Fig. 187).

AUSCULTATION

First sound

The first sound is markedly delayed in relation to the QRS complex which usually shows right bundle branch block. It is normal or diminished in intensity.

Second sound

The second sound is heard best at the lower left sternal border or the apex and is definitely diminished at the pulmonic area. In spite of the marked degree of right bundle branch block, the second sound at the pulmonic area was narrowly split in half of our patients and did not appear to be split at all in 3 others.

Third sound

A prominent third sound was present in all patients.

Fourth sound

Occasionally a fourth sound was present, but it was often masked by a presystolic murmur.

Systolic murmur

Every one of our patients had a systolic murmur of moderate *intensity* and medium frequency, either decrescendo or crescendo decrescendo in configuration, which was best recorded at the mid or lower left sternal border.

Presystolic murmur

Every one of our patients had a presystolic murmur which, because of the prolonged P R interval and the delayed *first sound*, was not crescendo in

encountered them in 10 per cent proved cases In two of these there was the rather uncommon anatomic arrangement of transposition of the great vessels associated with a ventricular septal defect and an excessive pulmonary outflow tract, together with a normal sized aortic outflow tract

Continuous murmurs were not encountered in any cases, despite the occasional presence of a small patent ductus arteriosus Of course, successful shunt operations result in the occurrence of typical continuous machinery murmurs (Fig 186)

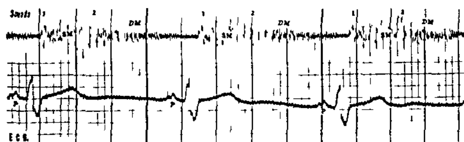


Fig 186 (2 L I S Log) Continuous murmur occurring after shunt operation for tricuspid atresia Note the normal intensity first sound (1) 0 10 sec after the onset of the QRS complex the second sound (2) not clearly distinguishable and the continuous systolic diastolic murmur (SM DM) maximum until about the time of the second sound and decrescendo throughout diastole This murmur is of high frequency and high intensity during systole but the intensity diminishes gradually throughout diastole

wave The second sound was diminished in intensity at the pulmonic area and was either narrow or normally split There was always a systolic murmur at the mid left sternal border and a presystolic murmur without crescendo at the lower left sternal border Apical mid-diastolic murmurs were recorded occasionally, and at times extended into presystole

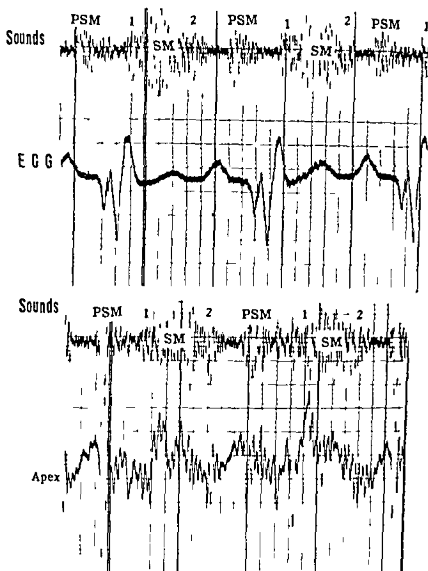


Fig 187 (Mid L S B Log with electrocardiogram and apex cardiogram) The mid diastolic presystolic murmur of Ebstein's anomaly Note the marked right bundle branch block and broad P wave in the electrocardiogram the normal first (1) and second (2) heart sounds the crescendo decrescendo systolic murmur (SM) and the diastolic presystolic murmur (PSM) of medium frequency and moderate intensity starting with accentuated vibrations which may represent a third heart sound

quality In some patients this murmur was comparatively intense and had a "scratchy" quality on auscultation

MID DIASTOLIC MURMUR AT THE APEX

Mid diastolic murmurs at the apex were not encountered frequently If present, they were of low intensity and medium to low frequency, and some times continued into presystole, but were not crescendic

SUMMARY

These patients had triple or quadruple rhythm at a relatively slow rate The first sound was normal in intensity but delayed in relation to the Q



Fig 189 (Apex Log) Atrial flutter and diastolic murmur in endocardial fibroelastosis Note the presence of atrial flutter waves (f) on the electrocardiogram the very loud first heart sound (1) and the loud second sound (2) the systolic murmur (SM) of low frequency and low intensity and the diastolic murmur (DM) of low frequency and medium intensity early in diastole

Endocardial fibro elastosis may be encountered during early infancy or, less commonly, in later life It is probably fair to say that if there is no history suggesting infectious myocarditis or paroxysmal tachycardia and if left to right shunts, hypertension, coarctation of the aorta, anemia, and aortic stenosis can be excluded, then, in acyanotic children beyond the age of 6 months who have cardiomegaly and congestive failure, endocardial fibro elastosis is the most likely diagnosis

Many of these patients have normal or quiet heart sounds, occasionally with a protodiastolic gallop, and no significant murmurs, however, there are other patients who have severe involvement of the mitral valve and who may have loud apical systolic mid diastolic, or presystolic murmurs (Figs 188 and 189) Frequently one encounters an association of coarctation of the aorta aortic stenosis and mitral valve involvement with the appropriate murmurs Recently we saw a 16 month old boy with severe coarctation of the aorta a tremendously enlarged heart, congestive heart failure, and ventricular hypertrophy We made a clinical diagnosis of coarctation of the aorta with endocardial fibro elastosis involving especially the left ventricle or the mitral valve At postmortem examination there was considerable involvement of the left atrium, and above the mitral valve there was a circular ridge of elastic tissue causing physiologic stenosis of the mitral orifice

29 / Myocardial Disease

Usually, myocardial lesions in children fall into one of four main groups

1 Infectious myocarditis (diphtheritic or viral myocarditis) Probably many of the so called "idiopathic" types of myocarditis will ultimately be included in this group
2 Metabolic myocardial lesions (glycogen storage disease of the heart)
3 Anatomic structural anomalies (anomalous origin of the left coronary artery from the pulmonary artery)
4 Myocardial lesions of unknown etiology (endocardial fibro elastosis)

Patients who have the left coronary artery arising from the pulmonary artery *or who have glycogen storage disease of the heart or medial necrosis* of the coronary arteries usually do not survive beyond the first few months of life The heart sounds are normal in intensity, and an apical third sound may be present If the rate is sufficiently rapid, these three sounds may give the effect of a protodiastolic gallop

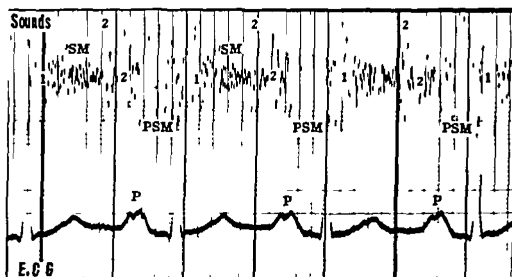


Fig 188 (Apex Log) Systolic and mid and late diastolic murmurs in a 4 month old boy with endocardial fibro elastosis Postmorten examination showed some involvement of the left ventricle but considerable involvement of the mitral valve with the leaflets fixed so that there was both *severe mitral stenosis and some degree of incompetence There was consider* able left atrial enlargement shown roentgenologically and at postmortem examination Note the P mitrale in the electrocardiogram the moderately loud first sound (1) 0 06 sec after the onset of the Q wave of the electrocardiogram the moderately intense second sound (2 2) split by 0 04 sec with the second part of the split more intense than the first the systolic murmur (SM) of moderate intensity and medium frequency and the mid diastolic presystolic murmur (PSM) of very high intensity and medium frequency

section VI

miscellaneous

30 / Arrhythmias

Variations from normal in cardiac rhythms are diagnosed much more accurately by the electrocardiogram than by any other method, our reason for including a chapter on the effect of various arrhythmias on heart sounds and murmurs is not because of any diagnostic triumphs we may hope to achieve but rather to emphasize certain interesting points and to mention some simple bedside tests which may facilitate the diagnosis by auscultation

NORMAL SINUS RHYTHM

Most children and adults with apparently normal hearts have resting heart rates varying between 60 and 100 beats per minute The rhythm is basically regular

Two heart sounds invariably accompany each normal cardiac cycle The first sound is caused principally by closure of the atrioventricular valves, and the second sound principally by closure of the semilunar valves In normal adults the first sound at the apex is almost always louder than the second sound at the apex but this statement does not apply in more than 50 per cent of children

SINUS ARRHYTHMIA

Sinus arrhythmia may be of several varieties, but the most common is characterized by changes in the length of the cardiac cycle occurring with respiration The heart rate tends to increase during early inspiration, as vagal tone diminishes, and slows during expiration, as vagal tone increases

Sinus arrhythmia is a normal finding in children and young adults but is encountered less commonly in older persons It is easily diagnosed by auscultation as the phasic variation in heart rate is typical Occasionally in older persons with slow rates the irregularity may be less characteristic and may be difficult to differentiate from that of atrial fibrillation or second degree heart block Exercise, the administration of atropine, or the development of fever increases the heart rate and abolishes sinus arrhythmia

Normally, the depolarization impulse originates in the sino atrial node and transmits throughout the heart, both the atria and the ventricles are equally involved and the P R interval is constant The arrhythmia is due to

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Normally, the depolarization impulse originates in the sino atrial node and transmits throughout the heart, both the atria and the ventricles are equally involved and the P R interval is constant The arrhythmia is due to

variations in vagal tone resulting principally from stimulation of pulmonary branches of the vagus nerve Drugs which increase vagal tone tend to produce or to accentuate sinus arrhythmia Digitalis is most important in this regard

SINUS BRADYCARDIA

By definition, sinus bradycardia implies a heart rate of fewer than 60 beats per minute, in normal persons the rate may drop to the low 40's without any ill effects Many well trained athletes have heart rates in the range of 45 to 55 beats per minute, but as age increases and physical fitness declines, the rate may increase to the accepted normal range

Levine and Harvey[1] reported a case of sinus bradycardia in which the heart rate of a completely healthy person without heart disease was 33 beats per minute Even at this slow rate careful measurement of the P R intervals by electrocardiogram may show sinus arrhythmia, but it is obvious that interpretation of the respiratory effect will be more difficult when there are only 2 or 3 cardiac beats with each respiratory cycle

Although the ventricular rate can be easily counted by precordial auscultation, it is imperative that a confirmatory electrocardiogram be taken to avoid overlooking heart block from various causes Careful clinical examination of all patients with sinus bradycardia is essential, as the possible causative diseases are many

Because of the large stroke volume necessary to maintain a normal cardiac output at a reduced heart rate, a vigorous pulsation may accompany certain of these disorders, and if such is the case, there may be accentuation of the first heart sound In other disorders, such as myxedema, the sounds may be diminished in intensity A low intensity third sound is often noted, and there may be a basal systolic murmur secondary to the increased stroke volume

SINUS TACHYCARDIA

In sinus tachycardia the heart rate is between 100 and 150 beats per minute, with a normal sinus origin for the cardiac impulse and normal conduction throughout the conducting system

The intensity of the heart sounds varies with the cause of the tachycardia, they may be weak following hemorrhage, or loud following severe exercise Auscultation rarely gives a clue to the underlying disease, but, conversely, a knowledge of the underlying disease may well explain the quality of the heart sounds

The shortening of diastole with the development of tachycardia frequently makes it difficult to distinguish the first from the second heart sound, but careful auscultation over the pulmonic area often permits identification of the sharper quality of the second sound The shortening of diastole with the

increase in rate also favors the production of a summation gallop, especially if the P R interval is prolonged

The differentiation between normal sinus tachycardia and tachycardia due to some abnormal mechanism should always be confirmed by an electrocardiogram Although it is usually true that sinus tachycardia slows gradually whereas abnormal tachycardias cease suddenly or in an irregular manner, this is not always the case

SINUS PAUSES OR SINUS ARREST

There may be complete dropping of one or several beats due to failure of the sinus node to discharge No electrocardiographic P wave results and no QRS follows in its normal sequence This is really a form of cardiac arrest and may persist for one or several beats Clinically, the patient may complain of the skipping of a beat or true Stokes Adams attacks may result

Auscultation is remarkable only in that there is complete disappearance of all heart sounds during the period of cardiac arrest and the first sound of the succeeding normal beat may be considerably accentuated

Sinus pause or sinus arrest also may occur secondary to digitalis toxicity in the presence of complete heart block when the ventricles continue to beat normally even though there may be periods of atrial standstill

HEART BLOCK

All forms of heart block may be temporary, intermittent, or permanent, and any one form may change into another or into normal sinus rhythm

Partial heart block

Our discussion of partial block will be concerned principally with heart block associated with sinus rhythm but it should be understood that a similar delay in transmission of the impulse through the atrioventricular node may occur with ectopic atrial pacemakers or with atrial flutter

Partial atrioventricular block is commonly divided into two main types —first and second degree block In first degree block there is abnormal prolongation of the P R interval, but each atrial impulse is followed by a ventricular contraction In second degree block there are more atrial than ventricular contractions over a given period but the ventricular contractions are stimulated by the atrial pacemaker

First degree block The tables of Ashman and Hull give the ranges of P R intervals for different heart rates prolongation of the P R interval beyond these normals constitutes first degree heart block The average range of the P R interval for heart rates between 60 and 100 beats *per minute* is from 0 12 to 0 20 sec

Auscultation In some patients with a prolongation of the P R interval

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increase in the P R interval from cycle to cycle, until finally the P wave is not followed by a QRS complex The cycle then begins again and is repeated The dropped ventricular complex may occur after 3, 4, 5, 6, or more beats and the number of complexes preceding a dropped beat is not necessarily constant Less frequently encountered is the Moebius type of second degree block, where the prolongation of the P R interval is constant, but occasionally the P wave is not followed by a QRS complex In each of these mechanisms the dropped beat is due to the fact that the P wave falls into the refractory period of the atrioventricular nodal conduction tissue, and the impulse fails to conduct

A third type of second degree heart block is sometimes seen in cases of paroxysmal atrial tachycardia secondary to digitalis toxicity There may be only one ventricular response to two or more atrial contractions A similar form of block is seen with atrial flutter and less commonly, with a normal sinus pacemaker (Fig 190)

Auscultation On auscultation, in all forms of second degree block with a regular ventricular response the effect on the intensity of the first heart sound depends on the P R interval of the conducted beat and the duration of the preceding diastole Additional diastolic sounds may occur and, if so are similar to those occurring with complete heart block Summation gallops, presystolic gallops, or atrial sounds occurring at regular intervals throughout the diastolic pause may be encountered also

In most of the cases in which the Luciani Wenckebach phenomenon (Figs 191 192 and 193) is involved there is little to be gleaned from auscultation, but certain possibilities are obvious

1 As the P R interval increases from cycle to cycle there may be a corresponding diminution in the intensity of the first heart sound until finally a beat is dropped

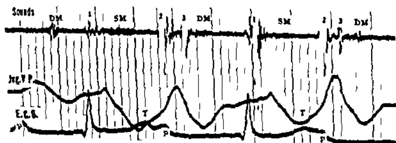


Fig 191 (Ap Log) An example of changing intensity of the first heart sound in Wenckebach's phenomenon Note the tremendous change in intensity of the first sound (1) from being relatively quiet in the first complex where the P R interval is 0 40 sec to being markedly accentuated in the second cycle where the P R interval is 0 49 sec Note also the systolic murmur (SM) of low intensity and medium frequency The second sound (2) is constant in intensity as is the third sound (3) Following each P wave there is a low intensity medium frequency diastolic murmur (DM) which [illegible] from late diastole in the first cycle to early diastole in the last two The maximal intensity of the first diastolic murmur occurs 0 22 sec after the onset of the P wave

the first heart sound is diminished in intensity This diminution probably results from the fact that the atrioventricular valve leaflets have floated up to an almost completely closed position prior to the onset of ventricular contraction

Sometimes in these patients the phonocardiogram may reveal a diminution in the intensity and frequency of the vibrations preceding the atrioventricular valvular component of the first heart sound It is not believed that the lessening of these vibrations plays any part in the diminished intensity of the first heart sound At this time it is worth noting that vibrations preceding the atrioventricular valve closure may be present in patients with atrial fibrillation, suggesting that atrial contraction alone is not responsible for them

The second heart sound is not altered in first degree block, but additional diastolic sounds may be heard, depending on the length of diastole and the length of the P R interval Atrial sounds may be heard individually, or a summation gallop may result from accentuation of a normal third heart sound by atrial systole

In patients with mitral presystolic murmurs, an increase in the P R interval causes the murmur to shift back from the first sound and to occur late in diastole rather than in presystole, giving a late diastolic crescendo decrescendo effect Occasionally, atrial systolic murmurs occur in patients with slow heart rates and first degree block, but with no obvious disease of the mitral valve

SECOND DEGREE BLOCK Depending on the ventricular response to the atrial pacemaker, second degree block may be associated with a regular or an irregular ventricular rhythm and with a normal or a slow ventricular rate If associated with supraventricular tachycardia or atrial flutter, the ventricular rate may be faster than normal Perhaps the best known form of second degree block is the Wenckebach type, where there is a progressive



Fig 190 (Apex Steth) Atrial contractions causing presystolic systolic and diastolic sounds in 3 1 heart block Note the normal intensity first sound (1) 0 05 sec after the onset of the QRS complex the normal second sound (2) and atrial sounds (4) following each P wave A low frequency vibration commences with the peak of the P wave and the maximal vibrations occur 0 12 sec after the onset of the P These atrial sounds are seen during the late part of systole mid diastole and presystole They are less intense during ventricular systole than during ventricular diastole

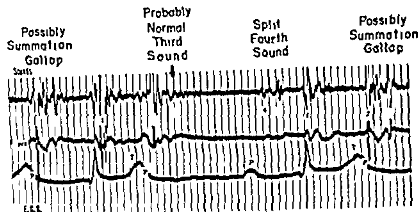


Fig 194 (Apex Log) Complete heart block showing variations in intensity of the heart sounds depending on the timing of atrial systole Note that the first sound (1) registered in the first complex is loud and follows a P R interval of approximately 0 48 sec In the second cycle the first sound is relatively normal and the P R interval is 0 37 sec

The second sound (2) is loud in all cycles and although its intensity does not vary greatly it is loudest in the first and third recordings when the peak of the P wave precedes it by 0 05 sec It is slightly less intense in the second cycle when the peak of the P wave precedes it by 0 10 sec

All the diastolic sounds have been labeled 4 on the assumption that they are caused principally by atrial systole The first two labeled 4 and the last sound labeled 4 occur early in diastole and vary greatly in intensity The peaks of the first and last P waves occur approximately 0 05 sec before the second sounds in these cycles and may aid early diastolic filling The peak of the second P wave occurs 0 10 sec before the second sound and atrial systole probably occurs during the late part of ventricular systole in this cycle This would account for the diminished intensity of the early diastolic sound (4) which however is probably a true third sound

Following the third P wave is a widely split sound (4) split by 0 09 sec occurring late in diastole It is possible but by no means certain that the first part of this split occurring 0 08 sec after the peak of the P wave represents atrial contraction and that the second part is created in the ventricular chamber either by the ventricular muscle or by the atrioventricular valves A further possibility is that the two components represent first right and then the left atrial contractions

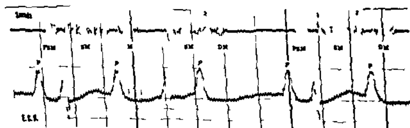


Fig 195 (Apex Log) Complete heart block with endocardial fibroelastosis and digitalis toxicity Note the first sound (1) of normal intensity 0 06 sec *after the onset of the QRS complex* and slightly less than in the third than in the first cycle even though the P R interval in each is 0 20 sec *Note also the* holosystolic murmur (SM) of moderate intensity and high frequency and the normal second sound (2) *Following the* tremendous P waves of the electrocardiogram are murmurs labeled DM or PSM depending on whether they lie in diastole or presystole These murmurs were *loud when the P wave occurred early in diastole* giving added impetus to early diastolic filling and were least when the P wave fell late in systole or immediately prior to *the QRS complex*

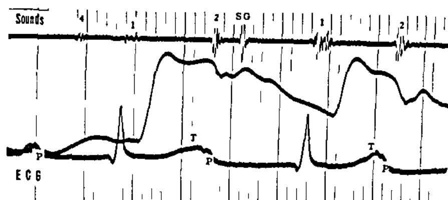


Fig 192 (Apex Log) Variations in heart sound with alterations of P R interval Note the first sound (1) of low intensity in the first cycle and of moderate intensity in the second cycle The first P R interval is 0 40 sec and the second is 0 44 sec The second sound (2) is normal in intensity in both cycles The fourth sound (4) in the first cycle occurs 0 22 sec after the onset of the P wave A summation gallop (SG) occurs early in diastole following the first complete complex This sound occurs 0 14 sec after the peak of the P wave and is almost certainly due to early diastolic ventricular filling accelerated by atrial systole

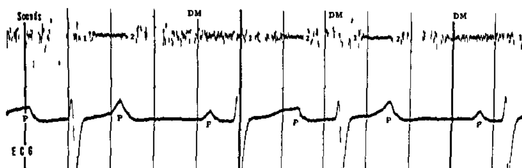


Fig 193 (4 L I S Log) Wenckebach s phenomenon showing the effect of the length of diastole on a diastolic murmur The rhythm is an example of Wenckebach s phenomenon with every third beat dropped There is a normal intensity first sound (1) 0 08 sec after the onset of the QRS complex and also a normal intensity second sound (2) Note the high frequency diastolic murmur (DM) This murmur is of high intensity early in diastole and in the long cycles it is decrescendo through to the succeeding first sound in the short cycles the murmur is of high intensity throughout diastole

2 If the atrial contraction is audible one may encounter successively a presystolic mesodiastolic protodiastolic or a summation gallop

Complete or Third Degree Atrioventricular Block

In complete or third degree atrioventricular block (Figs 194, 195, and 196), the atria and the ventricles are controlled by completely independ ent pacemakers The atria are commonly activated from the sinus node and beat at a rate of 70 to 80 beats per minute, showing normal sinus ar rhythmia Occasionally atrial flutter or atrial fibrillation may be present The ventricles are usually controlled by a pacemaker in the atrioventricular node or in the upper portions of the bundle of His, and they beat at a rate of 30 to 50 beats per minute, occasionally dropping to 15 and rarely exceed

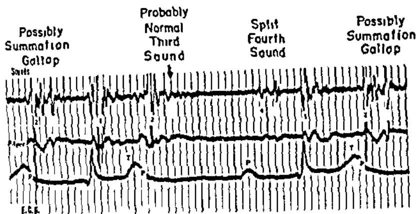


Fig 194 (Apex Log) Complete heart block show g variat ons n ntensity of the heart sounds dep nd ng on the t m ng of atr al systole Note that the F st sound (1) reg stered in the first complex s loud and follows a P R interval at approx mately 0 48 sec In the second cycle the first so nd s lat ely normal and the P R inter al is 0 37 sec

The second sound (2) s loud in all cycles and although its intens ty does not vary greatly t is lo dest n the first and thi d record ngs when the peak of the P wave precedes it by 0 05 sec It is sl ghtly less intens in the seco d cycle when the peak of the P wave precedes it by 0 10 sec

All the d astol c so nds ha e been labeled 4 on the ass mpt on that they are caused pr ncipally by at al systole The f st two label d 4 and the last sound labeled 4 occur early in diastole and ary g eatly in intens ty The peaks of the first and last P waves occur approximat ly 0 05 sec befo e the second sounds in th se cycles and may a d early d astol c fill ng The peak of the seco d P wa e occu s 0 10 sec befo e the second sound and atrial systole probably occ s d g the late part of ventricular systol n th s cycle Th s wo ld acco nt for the d m nished inte sity of the early d astolic sound (4) which however is prob bly a t ue th rd sound

F llow ng the th rd P wa e s a w dely spl t sound (4) spl t by 0 09 sec occurr ng late in d astole It s poss ble but by no means ce ta n that the first part of th s spl t occu ing 0 08 sec after th peak of the P wa e repr sents at ial cont act on and that the *second part is* creat d the v ntr cul ch mber e the by the vent cul r muscle or by the at io entr cular v l es A fu the poss b l ty is that the two components r present first r ght and then the l ft at al ont act ons

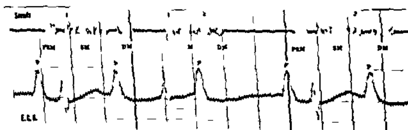


Fig 195 (Apex L g) C mpl t heart block with docard al fibro elastos s and d gital s to city Note the f st sou *d (1) of no m l* t s ty 0 06 sec after the ons t of the QRS c mplex d sl ghtly less t ns the th d than in the first cycle even though the P R n te al n e ch is 0 20 sec Note also the holosystol c *murmu (SM)* of mode ate intensity and h gh fr quency and th no mal second sound (2) *Follow* g the tremendous P waves of the electroca d g am are mu murs l b led DM or PSM depend g on whether they l e in d stole or presystole These murmurs w *loudest* when the P wa occu red ea ly in diastole g ng dded impetus to early diastol *c filling and* were l ast when the P wave fell late in systole or immed at ly p or to the QRS complex

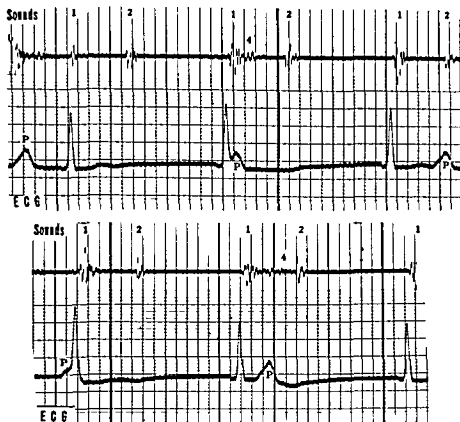


Fig 196 (Apex Log) Complete atrioventricular dissociation The atrial rate was 80 beats per minute and the ventricular rate 110 These tracings show some of the effects of varying time relations of the P waves and the QRS complexes on the first sound and on systole

Upper tracing In the first complex the P R interval equals 0 20 sec A few low intensity low frequency vibrations are present and the first sound consists of a narrow low intensity vibration 0 04 sec after the onset of the Q wave In the second complex the P wave falls just after the QRS complex the first sound is of normal intensity 0 04 sec after the onset of the Q wave and is followed by a group of low intensity low frequency vibrations (4) giving a systolic pseudosplitting of the first sound

Lower tracing In the first complex the P wave falls on the ascending limb of the R wave and the first sound is of normal intensity with systole being quiet In the second complex the P wave falls in mid systole causing a series of low frequency low intensity systolic vibrations (4) resembling a low intensity systolic murmur

ing 75, although we have one child with a rate of 120 beats per minute (Fig 197) In most instances, the duration of the QRS complex is normal, but if an idioventricular focus exists or if there is bundle branch block, the QRS duration will be lengthened

Auscultation Considerable variation in the intensity of the heart sounds can usually be detected on routine auscultation, but phonocardiograms, together with reference tracings (electrocardiogram and venous pulse tracing) are essential for the appreciation of lesser changes in intensity, variability in configuration, and the relationship of these observations to the timing of atrial and ventricular systole

First Heart Sound The first sound may vary in intensity from cycle to cycle, or it may be of constant intensity for several cycles and then appear

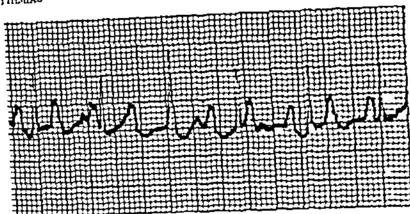


Fig 197 (E.C.G.) Complete heart block The atrial rate was 200 beats per minute and the ventricular rate 120

split or markedly accentuated In general, it may be stated that if atrial systole precedes ventricular systole by a normal interval (P R, 0 10 to 0 20 sec), the first sound will be of normal intensity If the P R interval is between 0 20 and 0 30 sec , the first sound will diminish whereas if the P R interval is more than 0 30 sec or less than 0 12 sec , the first sound will be accentuated

1 Bruit de canon In patients in whom atrial systole coincides with, or immediately precedes, the onset of ventricular systole, an extremely loud sound may result This sound was first described by Straschesko[3] in 1906 and given the name of 'bruit de canon ' Clarac and Pezzi,[4] in 1913, suspected the valvular origin of this accentuated sound

According to Lian and Bergamo,[5] the essential factor for the production of the canon sound is the firm fixation of the atrioventricular fibrous ring a rigidity which permits the atrioventricular valves to be firmly held between the chordae and the papillary muscles, on the one hand and their point of fixation on the other When the fibrous ring is not sufficiently fixed and the valves are relaxed less rapid and less frequent vibrations result When the P R interval is of normal duration the fixity of the atrioventricular ring is sufficient to give a distinct sound neither accentuated nor diminished If the P R interval is short or if the P and the R waves coincide, both atrial and ventricular systole will coincide with a greater fixity of the atrioventricular ring The same result will occur if the P wave immediately follows the R wave It is assumed that when the P R interval is greatly prolonged the relaxed atrium will have had time to refill and so add to the firm fixity of the atrioventricular ring This last statement is much less convincing than the others

Phonocardiograms of the first sound in complete heart block show obvious changes when a canon sound is produced There is a marked increase in the amplitude and frequency of the vibrations, as well as in the duration of the

entire sound A study of the initial vibrations of the first sound (often called the atrial vibrations) from beat to beat shows little change, irrespective of the P-R interval This suggests that these vibrations are certainly not entirely due to atrial systole Duchosal[6] described a "double canon sound" in a patient with bundle branch block and complete atrioventricular block It is assumed that this was due to separate sounds being formed at the asynchronous mitral and tricuspid valves

2 *Pseudosplitting of the first sound* When atrial systole occurs just before or just after the onset of ventricular systole, apparent splitting of the first sound may occur If atrial systole precedes the true first sound, a presystolic pseudosplitting may occur, whereas if an audible atrial sound immediately follows the first sound, systolic pseudosplitting results

The differentiation of pseudosplitting of the first sound from true splitting can be made easily on auscultation True splitting is independent of atrial systole and the P R interval and is consistent from beat to beat In contrast, pseudosplitting occurs only when the P-R interval is short, therefore it occurs only occasionally

3 *Diagnostic clues* The variable intensity and splitting of the first sound may be helpful diagnostically when the ventricular rate is normal Normally, patients with ventricular rates of 60 to 70 beats per minute would not be suspected of having complete atrioventricular block, and alterations in the intensity, and less commonly the presence of intermittent pseudosplitting of the first sound may be the only clues on auscultation In contrast to this, a normal first sound may be quite misleading in patients with atrial fibrillation and complete block This is encountered fairly frequently in patients who have received prolonged digitalis therapy here the intensity and splitting of the first sound do not vary from beat to beat

Second heart sound The second sound in complete atrioventricular block does not show the same dramatic changes in intensity as are noted in the first sound, but occasionally it may be considerably accentuated Pseudo splitting of the second sound may occur and may be either systolic or protodiastolic, in either case, the splitting is intermittent, depending on the timing of atrial systole

In 1937 Calo[7] pointed out that mechanical systole is prolonged in complete atrioventricular block in that *the second sound is delayed in relation to the onset of the QRS complex* He also noted that electrical activity of the ventricles is greatly prolonged and in fact the second sound occurs unusually early in relation to the end of the T wave These observations were confirmed by Lian, Marchal and Welti,[8] in 1938

Third Heart Sound Apical third heart sounds varying in intensity from beat to beat are usually encountered in patients who have complete atrioventricular block The intensity of the third sound varies from complete absence to a loud sound in those cases where the normal early rapid ventricular filling is assisted by atrial systole, and a summation gallop results

Atrial Sounds In complete heart block atrial sounds may be heard throughout the entire cardiac cycle The sounds may be relatively constant in intensity, although they are usually louder in diastole than in systole, they may be louder in early than in late diastole or they may be heard only at the time of the expected normal third sound, when a summation of effects may occur

The sounds are heard best at the apical and xiphoid regions, they are frequently low in intensity and have a somewhat distant quality (the "systoles en echo of Huchard) [9] Since there is no constant relationship between the atrial and ventricular contractions from cycle to cycle, the timing of the atrial sounds in relation to the first and second heart sounds varies, and a summation gallop if present, occurs intermittently, just as does the 'bruit de canon' of the first sound

Atrial sounds may be heard over the great veins of the neck, as noted by Josue and Godlewsky [10] in 1912, and can constantly be recorded in the esophagus [11]

1 During ventricular diastole As a general rule, atrial sounds or murmurs occurring in complete heart block are more intense during the early part of diastole than during the latter part This fact is explained by the greater blood flow into the ventricular cavity early in diastole and the lessening of the flow late in diastole, when the ventricular chamber has already filled Although this reasoning seems satisfactory, it is not always in accordance with the observed facts sometimes loud atrial sounds occur late in diastole

Tracings taken over the left atrium by intra-esophageal recording methods and in the right atrium and the right ventricle using a cardiac catheter reveal three groups of vibrations associated with atrial contractions recorded during ventricular diastole

The first group appears 0 04 to 0 06 sec after the onset of the P wave the second group from 0 06 to 0 15 sec after the onset of the P wave and the third group from 0 15 to 0 20 sec after the onset of the P wave (that is after the completion of atrial systole)

The first group usually appears during atrial systole and is recorded best by a right atrial catheter or an esophageal recorder The second group has been recorded by intra atrial intraventricular and precordial methods whereas the third group appears in intraventricular but not in intra atrial tracings Both the second and third groups are recorded on precordial tracings but the first group is not

2 During ventricular systole Atrial sounds are much less frequently recorded during ventricular systole than during ventricular diastole This can be explained simply by the fact that systole is shorter than diastole and also by the fact that during systole the atrioventricular valves are shut and so prevent blood flow into the ventricular chambers These two facts also explain the absence of atrial systolic murmurs during ventricular systole Another factor which may mask the atrial sounds in complete heart block is

the presence of apical or left sternal border systolic murmurs, which often have led to an erroneous diagnosis of ventricular septal defect.

Atrial sounds are not recorded during systole in all cases of complete atrioventricular block, and the intensity of these sounds varies from patient to patient. The atrial sounds recorded during ventricular diastole are usually louder than those recorded during ventricular systole, but occasionally the reverse is true.

3. *During ventricular fibrillation.* Because of the weak, incomplete contractions during ventricular fibrillation, the ventricles are unable to empty and become distended with blood. The atria continue to beat at their normal rate, and the atrial sounds may be audible. This fact was first noted by Clark and his associates in 1858.[12]

Systolic and diastolic murmurs. Systolic murmurs are almost always present with complete atrioventricular block. They are heard best at the apex, along the left sternal border, and also over the base of the heart. In congenital atrioventricular block, the presence of these murmurs has persistently contributed to the diagnosis of atrial or ventricular septal defects, while in adults mitral or tricuspid insufficiency has been suspected. Clarac and Pezzi[13] studied these murmurs in 1914. They noted their presence in most patients having atrioventricular block, they appreciated the variations in intensity and duration from beat to beat, and they attributed the cause to improper closure of the atrioventricular valves following the long P-R interval, with resulting mitral insufficiency.

Another factor in the causation of these murmurs may be of some significance. For the heart to maintain a normal output at a slow rate, stroke output must be increased and so must the diastolic volume of all the cardiac chambers, with some degree of generalized dilatation and, perhaps, stretching of the mitral and tricuspid valve ring, with true mitral and tricuspid insufficiency.

The diastolic murmurs in complete heart block without valvular lesions usually follow the short atrial sounds. They begin 0.14 to 0.23 sec. after the beginning of the P wave (and so after the completion of atrial systole). Since these murmurs appear in young children as well as in older persons, theories involving sclerosis of the atrioventricular valves in their causation must be dismissed or accepted only in part.

DIFFERENTIAL DIAGNOSIS. Complete heart block should be suspected immediately in any patient who has a slow ventricular rate and a regular ventricular rhythm. Careful auscultation will eliminate the other two common causes of a slow and regular ventricular rate, namely, sinus bradycardia and second degree heart block. In the former condition slow, deep breathing usually results in obvious sinus arrhythmia, there is no alteration in the intensity of the various heart sounds from cycle to cycle, and atrial sounds are not audible. In second degree heart block, atrial sounds may be audible

during ventricular diastole, but in 2 1, 3 1 or 4 1 heart block, the P R interval is constant, and there is no variability in the intensity of the heart sounds *from cycle to cycle* other than that due to normal respiration The one circumstance where heart sound intensity does not change in complete heart block is in the presence of atrial fibrillation Here the diagnosis can be completely overlooked by auscultation, and since the ventricular rate in patients who have been digitalized is frequently approximately 60 beats per minute, the diagnosis may not be even suspected

If the atrial rate is almost exactly double the ventricular rate there is little change in the P R interval from cycle to cycle, and a considerable period may elapse before any change in the intensity of the first sound can be noted Under these circumstances deep breathing, various forms of vagal stimulation, or exercise may sufficiently alter the atrial rate to produce more frequent alterations in the P R interval and the more frequent production of canon sounds

ECTOPIC BEATS

Atrial

Atrial premature beats (Figs 198 and 199) occur frequently in normal persons in children, especially they may be an early sign of digitalis toxicity In patients with heart disease atrial premature beats may be the forerunner of ectopic atrial tachycardia or atrial fibrillation

They may be encountered with shortened normal or prolonged P R intervals Frequently the associated first heart sound is accentuated, and it may be slightly delayed in relation to the Q wave of the electrocardiogram Split first sounds are rarely encountered with atrial ectopic beats, as the intraventricular conduction time is usually normal, and the *mitral and tri*

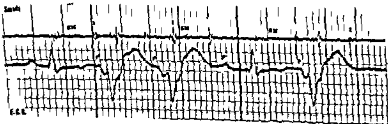


Fig 198 (Apex Log) Supraventricular premature beats occurring in a 14 year old girl who had no other cardiac abnormalities *With the normally conducted beats the first sound* (1) commences 0 05 sec after the onset *of the QRS complex* and is split by 0 02 sec with the first part of the split *of higher intensity and higher frequency than the second part* With the ectopic beats the first sound commences 0 05 sec *after the onset of the QRS complex* Wth both the sound is split by 0 03 to 0 04 sec and it is the second component which is of higher frequency and higher intensity *This pattern* is consistent throughout the tracing There is a low intensity medium frequency systolic murmur (SM) with the normally conducted beats this murmur is greatly reduced with the ectopic beats The second sound (2) is of normal intensity in all complexes Ventricular systole is shortened *with the ectopic beats*



Fig 199 (Apex Log) Wolff Parkinson White syndrome with a supraventricular ectopic beat Note the normal intensity first sound (1) 0 12 sec after the onset of the delta wave in the first and second beats It is accentuated after the ectopic beat and after the compensatory pause The accentuated first sound in the ectopic beat follows the Q wave by 0 11 sec and the delta wave in the last beat by 0 12 sec There is a low frequency low intensity systolic murmur (SM) Note the normal intensity second sound (2) following the first second and fourth beats and an accentuated second sound with the ectopic beat With the ectopic beat both electrical and mechanical systole are considerably shortened

cuspid valves shut almost simultaneously In ventricular ectopic beats, on the other hand, the intraventricular conduction time is prolonged, and asynchronous valve closure may result

Because of the short diastole preceding many atrial ectopic premature beats, there is diminished cardiac filling and possibly also reduced strength of ventricular ejection As a result, mechanical systole may be shortened, and the second heart sound may be diminished, or even absent

Ventricular

Ventricular ectopic beats (Fig 200) may occur with normal sinus rhythm or with atrial fibrillation Occasionally they occur after every one or two normal beats, resulting in bigeminy or trigeminy

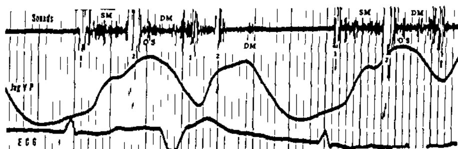


Fig 200 (Apex Log) Variations in the intensity of the heart sounds and murmurs in rheumatic heart disease with atrial fibrillation and ventricular ectopic beats The normally conducted complexes have a normal intensity first sound (1) 0 09 sec after the onset of the QRS complex a medium frequency moderate intensity systolic murmur (SM) an intense second sound (2) an opening snap (OS) and a long diastolic murmur (DM)

With the ectopic beat several changes occur The first sound (1) is diminished in intensity delayed 0 12 sec after the onset of the QRS complex and is widely split (0 06 sec) there is moderate reduction in the intensity of the systolic murmur and marked reduction in intensity of the diastolic murmur systole is greatly shortened and there is diminished intensity of the second sound

AUSCULTATION

On auscultation ectopic beats are recognized by the sudden interruption of a basically normal rhythm due to a beat which occurs earlier than normal and is followed by a prolonged pause If ventricular ectopic beats are frequent, it may be impossible to differentiate the rhythm from that of atrial fibrillation

FIRST AND SECOND SOUNDS The characteristics of both the first and the second heart sounds vary according to the site of origin of the ectopic beat and, more especially, with the timing of the ectopic beat in the cardiac cycle

The first heart sound is always delayed in relation to the onset of the QRS complex of the electrocardiogram because of (1) the abnormal pathway of ventricular depolarization, (2) the distance the atrioventricular valves must travel in order to close, and (3) the time required for the development of sufficient ventricular pressure to exceed atrial pressure

Frequently the first sound accompanying the ectopic beat exceeds the normal first sound in intensity This is due in part to the widely-opened position of the atrioventricular valves at the onset of ventricular systole, but it requires a ventricular contraction of normal or near normal intensity Since ventricular systoles resulting from impulses arriving during the partial refractory period of ventricular musculature result in weak contractions it follows that the first heart sound may be diminished in intensity or even in *audible* Occasionally, because of the abnormal depolarization pathway, as shown by the bundle branch block pattern, there may be splitting of the first heart sound Presumably this splitting is due to asynchronous closure of the atrioventricular valves, with the valve of the ventricle in which the beat originates closing first

As pointed out by Cossio,[14] "When the valves have further to travel between the onset of ventricular contraction and the position of maximum stretch, their movement is accelerated and consequently their vibration is increased for the kinetic energy is the product of half the mass and the square of the velocity, and the velocity is the product of acceleration and time"

In all forms of premature beats atrial nodal, or ventricular, the atrioventricular valves have a greater distance to travel before closure, this is even more marked in those beats preceded by a P wave, when the valve cusps are forced even further down into the *ventricular* cavity A loud first sound is most likely to follow an ectopic beat when such a beat falls in mid diastole after the ventricular muscle has completely recovered from its previous systole and while the atrioventricular valves are still well down in the ventricular cavity

Sometimes atrial premature beats are not followed by ventricular contractions it is not possible to make this diagnosis by auscultation, as there is simply an absence of heart sounds with the dropped beat

Frequently, the second heart sound is split because of asynchronous closure of the semilunar valves secondary to the delayed conduction time The intensity of the second sound depends on the force of ventricular systole, which determines the resulting cardiac output and the aortic and pulmonary artery pressures Often, if the ventricular contraction is weak, the semilunar valves do not open, and no second sound is audible When the semilunar valves do open, mechanical systole is commonly shortened in comparison with electrical systole, and the second sound occurs earlier than usual If systolic murmurs are present with normal sinus beats, they may be diminished or absent with the ectopic beats It is extremely rare for a murmur not previously present to be generated by the ectopic beat The murmurs of both aortic and pulmonary insufficiency are diminished, secondary to the lowered pressure in the aortic and pulmonary arteries associated with ectopic beats, and if the beats fall early enough, mid diastolic murmurs, from whatever cause, will be obliterated from the diastole of the cycle preceding the ectopic beat For obvious reasons, atrial gallop sounds and presystolic murmurs due to atrial systole are not audible with the ectopic beat

Relationship of the First and Second Heart Sounds to Cycle Length 1 *First sound* As a general rule, in most arrhythmias, it may be stated that the intensity of the first heart sound tends to vary inversely as the duration of the preceding diastole This statement is explained by assuming that if diastole is short, the atrioventricular valves will be wide open and will have to travel some distance before being closed, in contrast, if diastole is prolonged, the valves will have floated up to an almost completely closed position before the onset of ventricular systole, and a slight tensing of the valves will be all that is needed to complete their closure There are many exceptions to this rule, since it is obvious that if ventricular systole is sufficiently weakened the first sound may be diminished or even absent

2 *Second sound* The second sound varies in the opposite manner to the first sound If diastole is extremely short, the ventricular beat is often weak and the volume of blood in the ventricular cavity being less than normal, the stroke output is diminished This may result in complete inability of the ventricular systole to open the semilunar valves or, if the valves do open the aortic pressure may be diminished and, also, the intensity of the second heart sound

Ectopic premature beats

Ectopic premature beats may be atrial, nodal, or ventricular in origin and may occur in almost any form of heart disease or in normal persons The ectopic beats may be infrequent and, apart from causing a consciousness of some irregularity of rhythm, may be quite innocuous In other instances ectopic beats may be the forerunners of more severe and permanent irregularities

Atrial and ventricular premature beats are frequently diagnosed during auscultation, but atrial premature beats cannot be separated clinically from nodal premature beats, and, although ventricular premature beats can usually be distinguished by the compensatory pause which follows, it is well to realize that a similar pause also may occur following atrial premature beats

If ectopic beats occur with sufficient force, two heart sounds will result, but if the beat is weak, as often happens when the ectopic beat begins in the early part of diastole or when there are multiple ectopic beats, it may be extremely difficult to hear one or both heart sounds

BEAT FOLLOWING THE PREMATURE BEAT

Because the ventricles have a longer rest period than normal following a premature beat, it is not surprising that the succeeding beat often is stronger than the normal beat As a result the first and second sounds are frequently accentuated, as also are any associated systolic murmurs Presystolic murmurs normally associated with mitral and tricuspid stenosis are often absent The obvious explanation would seem to be that the ventricles are filled after the prolonged diastole, and atrial systole is unable to contribute enough additional flow to cause turbulence and so generate an audible murmur

Ventricular extrasystole may modify not only the heart sounds and murmurs of the cycle following the abnormal electrocardiographic impulse but also those occurring during the diastole of the previous cycle, together with those of one or more succeeding cycles

NODAL RHYTHM

The first heart sound may change *in intensity* with the onset of nodal *rhythm* Depending on the relationship of the P wave to the QRS complex of the electrocardiogram the first sound may be *normal*, *increased* in intensity or split When the P wave falls just prior to the onset of the QRS complex, atrial contraction may force the atrioventricular valves down into the cavity and the increased force of closure may result in an accentuated first sound or presystolic pseudosplitting *of the first* sound If the P wave is hidden in the QRS *complex* the first sound *may be* of normal intensity, but there may be a diminution in the number and the intensity of the vibrations preceding the closure of the atrioventricular valves (that is a reduction in the so called 'atrial *component*' of the first sound)

Where atrial contraction follows the QRS complex and the closure of the atrioventricular valves an atrial sound may be heard in the early part of ventricular systole, *giving the impression of splitting* of the first sound (Fig 201) (systolic pseudosplitting)

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Ectopic premature beats

Ectopic premature beats may be atrial, nodal, or ventricular in origin and may occur in almost any form of heart disease or in normal persons The ectopic beats may be infrequent and, apart from causing a consciousness of some irregularity of rhythm, may be quite innocuous In other instances ectopic beats may be the forerunners of more severe and permanent irregularities

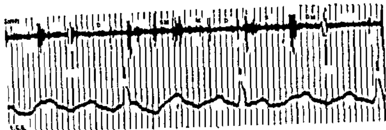


Fig 202 (Apex Log) Atrial flutter with quadruple rhythm due to a protodiastolic summation sound and a presystolic sound in the first and third cycles In the second cycle the flutter wave falls slightly later in relation to the third sound so that a summation does not occur and the third sound appears split also the presystolic sound is nearer the succeeding first sound Note the normal intensity first sound (1) 0 07 sec after the onset of the QRS complex the systolic murmur (SM) of low intensity and medium frequency the normal intensity second sound (2) and the atrial sounds (f) of variable intensity as described above always noticeable during ventricular diastole and occasionally during ventricular systole

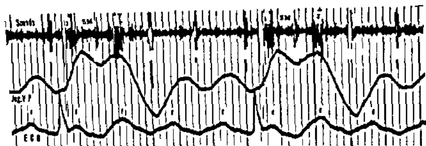


Fig 203 (Apex Log with simultaneous jugular venous pulse tracing and electrocardiogram) Atrial flutter with atrial sounds occurring during both ventricular systole and ventricular diastole Note the loud split first sound (1) split by 0 03 sec The first part of the split occurring 0 07 sec after the onset of the QRS complex is much louder than the second component which follows the QRS by 0 10 sec The second sound (2) is normal Multiple atrial sounds (4) follow the summit of the P waves by approximately 0 16 sec The intensity of the atrial sounds varies with the r p sition in the cardiac cycle Those occurring during ventricular systole are less intense than those during ventricular diastole Of the atrial sounds occurring during ventricular diastole the loudest are early in diastole and probably represent a summation gallop The atrial sounds in presystole are louder than those in mid diastole The atrial sounds always occur toward the trough in the jugular venous pulse tracing This occurs during both ventricular systole and ventricular diastole the significance of this is not entirely clear

sounds may be heard throughout the cardiac cycle, occurring during ventricular systole ventricular diastole, or both

It may not be possible to distinguish atrial flutter with a changing block from atrial fibrillation or from paroxysmal atrial tachycardia with varying atrioventricular block

In patients with atrial flutter and varying block, the diagnosis sometimes may be clarified by having the patient exercise, a constant 2 1 or 1 1 flutter may result with a steady ventricular rate of 150 or 300 beats per minute Unfortunately many patients are in no condition to exercise

Another helpful bedside test may be vagal stimulation In sinus tachy

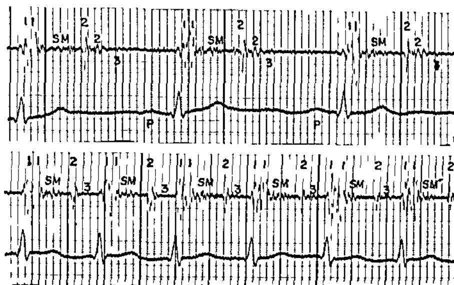


Fig 201 (Apex Steth) Systolic pseudosplitting of the first heart sound with nodal rhythm in an 18 year old white male in excellent health Cardiac rhythm at rest varies between normal sinus sinus bradycardia nodal tachycardia and paroxysms of atrial tachycardia With exercise the rhythm always becomes normal sinus

Upper tracing (normal sinus rhythm) Note the narrowly split (0 02 sec) first sound (1 1) terminating 0 10 sec after the onset of the Q wave the low frequency low intensity systolic vibrations (SM) the well split (0 06 sec) second sound (^ 2) and the very low intensity low frequency third sound (3)

Lower tracing (nodal tachycardia) Note the well split (0 04 sec) first sound (1 1) terminating 0 13 sec after the onset of the Q wave The delayed second part of this apparent split may be due to the delayed atrial systole secondary to the nodal tachycardia giving systolic pseudosplitting of the first sound There is no significant change in the systolic murmur (SM) the second sound (2) or the third sound (3)

ATRIAL FLUTTER (FIGS 202 AND 203)

In atrial flutter the atria beat regularly at a rate of 300 ± 50 beats per minute If quinidine is administered, the rate may be reduced to less than 200 beats per minute

Varying degrees of heart block may occur with atrial flutter, and the degree of block may change frequently

Assuming an atrial rate of 300 beats per minute,

1 1 2 1 3 1 4 1	flutter gives a ventricular rate of	300 150 100 75	beats per minute

Two to one block with a ventricular rate of 150 beats per minute may easily be mistaken for sinus, atrial, or nodal tachycardia, and 4 1 block may pass completely unsuspected

If the heart is relatively normal, apart from the rhythm, no atrial sounds can be heard, but if atrial hypertrophy exists for any reason, atrial flutter

TACHYCARDIAS

In considering any severe prolonged tachycardia it must be appreciated that murmurs which are frequently heard may be secondary to the cardiac dilatation which often accompanies such tachycardias, and one must be careful in attributing these murmurs to organic valve damage

It is important to realize that with increasing heart rates diastole shortens much more than systole and that, while at rates of 150, systole and diastole are approximately equal yet at rates of 200, the systolic interval considerably exceeds the diastolic This often makes it difficult to distinguish the first heart sound from the second on auscultation, and if any additional sounds are present, a phonocardiogram may be essential for intelligent interpretation

Effect of tachycardias on murmurs associated with valvular lesions

During attacks of tachycardia it is a common experience to notice a diminution of cardiac murmurs associated with valvular lesions This applies especially to diastolic murmurs, both basal and apical, and to a lesser extent, to systolic murmurs The accepted explanation of this diminution in intensity of previously well recognized murmurs is the diminished flow across the valve orifices during each cardiac cycle

Paroxysmal atrial tachycardia (Figs 204 and 205)

In adults with paroxysmal atrial tachycardia the heart rate usually lies between 160 and 190 beats per minute, although rates of more than 200 or less than 150 may be encountered The onset is sudden and the rhythm is usually perfectly regular The rate may slow considerably during sleep and minor variations in rate may occur over periods of several hours

The heart sounds are normal for the rate because of the shortened diastole it may be difficult to differentiate between the first and second sounds

With second degree atrioventricular heart block Paroxysmal atrial tachycardia with second degree atrioventricular heart block is sometimes encountered as a result of digitalis toxicity and may also occur without obvious cause On auscultation this arrhythmia may resemble any of the other rapid irregular rhythms depending on the degree and the constancy of the atrioventricular block It can be diagnosed easily by electrocardiogram

There may be considerable variation in the intensity of both the first and second heart sounds depending on the degree of atrioventricular block This same variation in heart sounds is encountered in atrial flutter with changing block and in atrial fibrillation with a rapid irregular ventricular rate The intensity of the first sound varies with the duration of the P R interval and the length of the preceding diastole

cardia there may be a temporary slowing and then a gradual return of the tachycardia after vagal stimulation is removed In paroxysmal atrial tachycardia, sudden complete reversal of the rhythm to normal may result, but the reversion may also occur gradually over a period of many beats In atrial flutter there may be a sudden temporary slowing of the rate, with an irregular jerky return to the rapid rate after vagal stimulation ceases

Atrial sounds are not usually audible in patients with atrial flutter but occasionally they may be fairly clear, and even striking The sounds may be heard throughout the entire cardiac cycle, but are usually louder in diastole than in systole and are frequently louder early rather than late in diastole As with atrial sounds in complete heart block, the flutter sounds occur after the flutter wave on the electrocardiogram and fall at the end of the down stroke of the jugular venous pulse wave

In the early part of diastole a loud sound may occur which is probably a summation gallop, the timing of this sound, together with the other diastolic flutter sounds can be explained easily by assuming that these sounds are produced in the ventricular muscle, in the atrioventricular valves, or in both As a result, one would expect to record these sounds after the electrocardiographic flutter waves and at the termination of the jugular venous pulse wave To explain the similar timing of these sounds during ventricular systole *is more difficult Here, presumably, the atrioventricular valves are* closed, and an atrial sound, if produced, could reasonably be expected to occur at the height of atrial contraction It is difficult to conceive of atrial contraction exerting enough force to open the atrioventricular valves except toward the end of systole, during the relaxation phase (Fig 203)

Flutter sounds may be heard at the cardiac apex, but are heard best at the base of the heart in the second and third interspaces, either to the left or to the right of the sternum The sounds are usually dull but may be comparatively loud Depending on the degree of atrioventricular block accompanying the flutter, triple or quadruple rhythms may be encountered, or several flutter sounds may be heard during ventricular diastole Depending on the relationship of the flutter sounds to the normal heart sounds, one may encounter presystolic, systolic, protodiastolic, or summation gallop, or pseudosplitting of the first or second heart sounds If changing atrioventricular block accompanies the flutter there may be a change in the intensity of the flutter sounds from cycle to cycle

Factors contributing to the production of flutter sounds appear to be (1) a good atrial contraction, (2) poor tone of the ventricular myocardium, and (3) a long diastolic pause The importance of the first is obvious from the fact that flutter sounds disappear with the onset of atrial fibrillation, the second and third factors would seem to be important since flutter sounds may disappear as clinical improvement results or if there is an increase in ventricular rate

VENTRICULAR TACHYCARDIA

In ventricular tachycardia the first sound may be of variable intensity Since the P waves occur at a much slower rate than the QRS complexes there is a variable relationship between atrial and ventricular contraction At times the atrial contraction may immediately precede the ventricular contraction, and true "canon" sounds may result, but throughout most of the tracing the first sound may be normal, slightly accentuated, or considerably diminished As with atrial flutter, or complete atrioventricular block, small atrial sounds may occur occasionally between the first and second heart sounds

The first sound will also be louder after a longer diastolic pause, this loud first sound is usually accompanied by a stronger peripheral pulse, emphasizing the importance of the strength of ventricular contraction in the production of this sound

Because of the considerable degree of intraventricular block commonly seen with ventricular tachycardia, there may be asynchronous closure of the atrioventricular valves of the semilunar valves, or of both As a result splitting of either the first or second sounds may occur

Because of the shortening of systole and diastole with the onset of the tachycardia, and because of the lower stroke volume and the lowered systemic pressure, ventricular tachycardia causes an alteration and diminution in the intensity of the systolic murmurs of aortic or pulmonic stenosis and the murmurs of aortic or pulmonic insufficiency There is also a diminution in murmurs associated with patent ductus arteriosus ventricular septal defects and other lesions associated with left to right shunts Conversely murmurs of mitral or tricuspid insufficiency may develop secondary to cardiac dilatation

ATRIAL FIBRILLATION

Atrial fibrillation is the most commonly encountered of the important arrhythmias and usually it is recognized easily by the total irregularity of the rhythm (Fig 206)

In 1913 Lewis[1] obtained the first phonocardiogram of atrial fibrillation He described the great variability in the intensity of the heart sounds and noted the disappearance of the presystolic murmur of mitral stenosis with the onset of atrial fibrillation In 1915 Battaerd[10] drew attention to the variable duration of systole and in 1930 Wolferth and Margolies[1] noted the variability in the intensity of the first sound Since the intervals between the ventricular contractions are irregular and the force of the contractions is variable it follows that the intensity of the accompanying heart sounds and murmurs will change from cycle to cycle As confirmation of the changing intensity of ventricular systole the radial pulse wave may vary in strength from cycle to cycle or there may be a disproportion between the

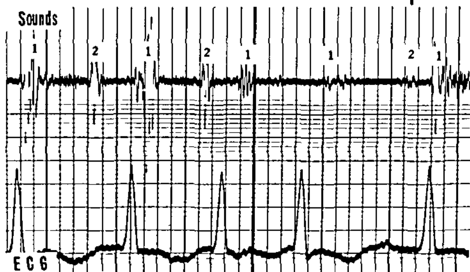


Fig 204 (Apex Log) Multifocal supraventricular tachycardia showing variation in the intensity at the first sound (1) and the second heart sound (2) Both the heart sounds are considerably diminished in intensity following a short diastole and the second sound is absent in the third cycle In the third and fourth cycles the onset of the first sound (1) is 0 09 sec after the onset of the QRS whereas it is not more than 0 05 sec in the other cycles The sound labeled 2 in the fourth cycle and marked by an arrow probably is not a true second sound but may be the result of atrial activity The poor quality and the delay of the first sound in this cycle suggests the possibility that the semilunar valves may not have opened There is no reason why systole should be prolonged in this cycle and such would have to be the case if this is a second sound

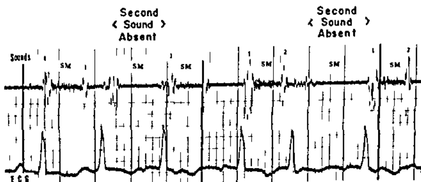


Fig 205 (Apex Log) Supraventricular tachycardia with an irregular ventricular rhythm and multiple foci of origin of the atrial complexes Note the variations in intensity of the first heart sound (1) which is louder following the longer pauses as in complexes three four and six The first sound is greatly diminished in the fifth complex following a short diastole There is no significant alteration in the time of onset of the first sound Following a short diastole the intensity of the systolic murmur diminishes considerably Variations in the intensity of the second sound (2) depend on the length of the previous diastole In the second and fifth cycles no second sound can be identified Either these beats did not occur with sufficient force to open the semilunar valves or if the valves did open the ejection pressure may have been minimal Low frequency low intensity vibrations occur before the main valvular component of the first sound in each cycle even though no typical P waves are seen

regular ventricular beat This occurs particularly in those patients who have been receiving digitalis for some time and in whom complete heart block develops with atrioventricular nodal rhythm Under these circumstances the heart sounds do not vary in intensity, there is no peripheral pulse deficit, and the condition cannot be diagnosed by auscultation

It is commonly stated that atrial fibrillation may easily be distinguished from frequent ventricular ectopic beats by having the patient exercise, during exercise, the fibrillation becomes more obvious, and ventricular ectopic beats disappear This test presumes that the patient will be well enough to perform physical exercise, but unfortunately this is not always the case

Multiple atrial premature beats paroxysmal atrial tachycardia with block and atrial flutter with changing block may all be confused clinically with atrial fibrillation and the electrocardiogram is by far the best method of distinguishing these arrhythmias

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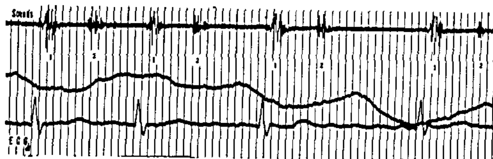


Fig 206 (Apex Log) Atrial fibrillation with increasing cycle length showing no essential change in the intensity of the first heart sound (1) or the second sound (2) Note the normal intensity first sound (1) split by 0 03 sec with the maximal vibrations 0 06 sec after the onset of the QRS complex and the low intensity second sound (2) split by 0 02 sec The second part of the split is loudest in the first cycle The first part of the split is loudest in the second cycle The two parts of the split are equal in the third cycle These minor changes were not identifiable on auscultation

number of apical beats and the number of palpable peripheral arterial pulse beats

Atrial sounds and murmurs

With the onset of atrial fibrillation all sounds of atrial activity, such as presystolic gallop and the classical presystolic crescendo murmurs of mitral stenosis, disappear

Diastolic sounds and murmurs

Because of the irregular occurrence of ventricular contractions in atrial fibrillation, the length of diastole varies from cycle to cycle and affects the clarity with which diastolic sounds and murmurs can be detected If diastole is long any sounds or murmurs can be easily detected However, if ventricular systole falls early in the diastole of the previous cycle, an otherwise long diastolic rumble becomes greatly shortened and may even sound crescendic If diastole is sufficiently short no diastolic murmurs can be heard at all

Intensity of the second sound and duration of systole

With those beats which occur early in diastole and fall in the partially refractory period of the ventricular musculature, the resulting stroke volume is diminished, systole is shortened, and the second sound occurs early and is diminished in intensity Similarly, any murmurs of aortic or pulmonic insufficiency become difficult to hear because of the lowered pressure in the aorta and in the pulmonary artery With most beats, systole is of normal or near normal strength, since except in beats occurring very early the early diastolic filling phase which accounts for the greater part of ventricular filling is not affected, and stroke volume is fairly constant

Occasionally atrial fibrillation may be accompanied by a fairly slow but

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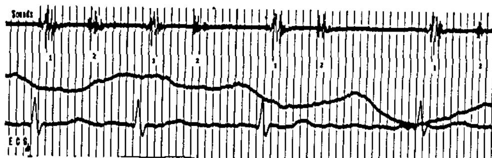


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31 / Early Diastolic Sound in Constrictive Pericarditis

The majority of patients with constrictive pericarditis have an early diastolic sound (Figs 207 and 208) This sound was first described by Potain[1] in 1856 In 1933, Lian and his co workers called it a "pericardial protodiastolic vibration" in order to distinguish it from a normal third heart sound or a protodiastolic gallop Eliasch and his associates,[3] in 1950, showed that the sound coincided with the nadir of the early diastolic dip in the right ventricular pressure curves of these patients McKusick,[4, 5] in 1952, using simultaneous phonocardiograms and ventricular border electrokymograms, demonstrated that the sound coincided with a sudden halt in rapid ventricular filling early in diastole

This sound is variable in intensity and may be louder than the first or the second heart sounds Frequently it is accompanied by a palpable apical thrust, also described by Potain[1] in 1856, it follows the beginning of the second sound by 0 09 to 0 13 sec

Differentiation from a split second sound is usually simple, as frequently a split second sound is present also The early diastolic sound is widely separated from the second sound, and, even when not loud it may be heard over a wide area These factors, in addition to its accentuation with inspiration, help to differentiate it from a normal third sound

The protodiastolic sound is not necessary for the diagnosis of constrictive pericarditis and although it is almost always present with calcification of the pericardium even then it is not essential It is helpful diagnostically in the absence of calcification and when the heart is larger than that normally encountered in this condition

The early diastolic sound in constrictive pericarditis is thought to be due to the water hammer phenomenon resulting from a sudden cessation of the ventricular early rapid filling phase caused by the rigid pericardium Although there is much interesting evidence to support this theory, it should be emphasized that the exact cause of the sound has not yet been proved

The right ventricular pressure tracings of patients with constrictive pericarditis usually show an early diastolic dip, and the protodiastolic sound occurs just beyond the nadir of this dip The dip reflects an abrupt fall of right ventricular pressure, and the rise which follows probably is due to ventricular filling

Mounsey[6] performed simultaneous cardiac catheterization and phono

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tricular pressure curve Since the beginning of the pressure rise in the right ventricle precedes that in the right atrium, superior vena cava, and innominate vein, it is assumed that the sudden cessation of rapid filling originates from a pressure increase in the right ventricle

In all 4 of Mounsey's patients, the early diastolic sound coincided closely with the beginning of the steep upstroke following the early diastolic dip and was, therefore, synchronous with the abrupt halting of early diastolic filling

Pressure tracings of patients with constrictive pericarditis who did not have the early diastolic sound showed a less abrupt upstroke following the early diastolic dip, and the upstroke merged smoothly into the diastolic plateau

Mounsey stated that the abrupt halting of ventricular expansion may be noted at the time of thoracotomy in patients who have loud early diastolic sounds

It would seem, if the previous discussion is true, that the extra sound should disappear or occur later and be less intense after removal of the pericardium This is usually the case after a successful operation, but in some patients the jugular venous pressure remains elevated and, either because of incomplete removal of the pericardium or because of associated endomyocarditis, there may be little change in the physiologic state of the patient Under these circumstances the sound persists

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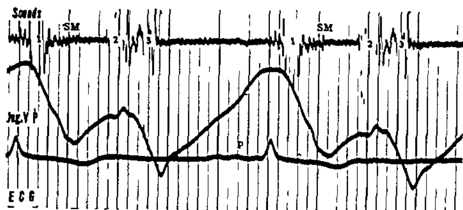


Fig 207 (Apex Log) Prominent early diastolic sound in constrictive pericarditis Note the loud first sound (1) with the maximal components occurring 0 10 sec after the onset of the Q wave the early systolic vibrations (SM) of low frequency and low intensity the loud second sound (2) split by 0 05 sec and the very loud third sound (3) 0 10 sec after the second sound This sound was easily palpable on clinical examination

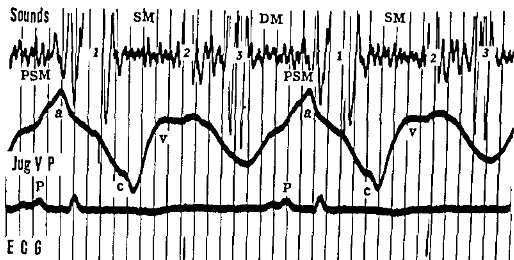


Fig 208 (Apex Steth with jugular venous pulse and electrocardiogram) A loud early diastolic sound a presystolic murmur and P mitrale in a patient with constrictive pericarditis Note the loud first sound (1) starting 0 05 sec after the onset of the Q wave the systolic murmur (SM) of low frequency and moderate intensity the very loud second sound (2) the markedly accentuated early diastolic sound (3) 0 12 sec after the end of the second sound and the presystolic crescendo murmur (PSM)

cardiography in 6 patients, and in the 4 in whom the right ventricle was entered, the nadir of the early diastolic dip in the right ventricle curve preceded the timing of a corresponding dip in the right atrial curve by 0 1 to 0 2 sec Examination of the pressures in the right heart chambers and in the peripheral veins showed a rapidly increasing pressure difference between the right ventricle and the basilic vein during the early part of the diastolic dip These pressure differences suggested that rapid ventricular filling takes place during the descending limb of the early diastolic dip and that filling continues but quickly decreases during the steep upstroke of the right ven

with long standing hypertension, congestive heart failure may develop at which time a protodiastolic gallop may be heard With ventricular failure, an increase in atrial pressure may also result in a presystolic gallop, giving a quadruple rhythm (Fig 209) Left ventricular dilatation is often accompanied by dilatation of the mitral ring, and the typical murmurs of mitral

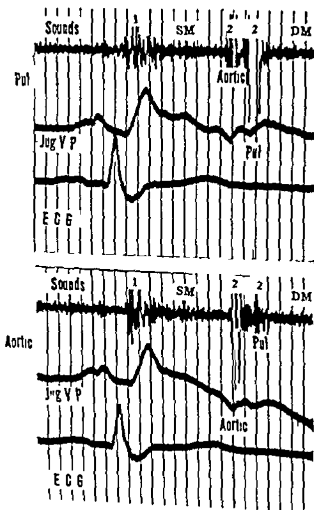


Fig 210 The relative intensities of pulmonic and aortic valve closure at the pulmonic and aortic areas respectively in a patient with essential hypertension with left ventricular failure and increased pulmonary artery pressure

Upper tracing (2 L I S Log) Note the normal first sound (1) 0 07 sec after the onset of the Q wave the systolic murmur (SM) of medium frequency and low to moderate intensity and the second sound (2 2) with the first or aortic component of normal intensity followed 0 06 sec later by a strikingly accentuated pulmonic closure

Lower tracing (2 R I S Log) The first sound (1) and the systolic murmur (SM) are essentially similar to those in the pulmonic area The second sound (2 2) is markedly altered The first or aortic component is now strikingly accentuated and is followed 0 06 sec later by a much less intense pulmonic component

32 / Systemic Arterial Hypertension

The diagnosis of arterial hypertension is made by the sphygmomanometer, auscultation neither confirms the diagnosis nor helps in differentiating the various causes Because of the common occurrence of this disorder, we shall mention briefly the auscultatory phenomena

AUSCULTATION

When hypertension is mild the heart sounds are normal and there are no significant murmurs If more severe, the first sound at the apex is normal or slightly accentuated, and there may be accentuation of the aortic valve closure at the second right interspace A grade 1 systolic murmur is commonly encountered at the aortic area This is probably due in part to the increased force of left ventricular ejection, in older patients it may be accentuated by the physical characteristics of an atherosclerotic aorta In younger patients with severe progressive hypertension and older patients

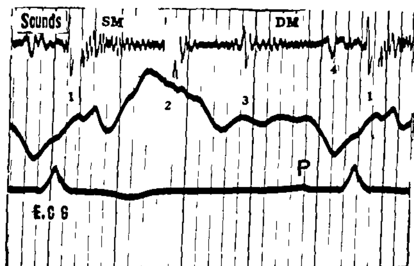


Fig 209 (Apex Log) Severe systemic hypertension and congestive failure in a 28 year old man showing a quadruple rhythm and a mid diastolic murmur Note the normal intensity first heart sound (1) 0 09 sec after the onset of the QRS complex the decrescendo systolic murmur (SM) of low intensity and medium frequency the prominent second sound (2) the well marked third heart sound (3) and fourth sound (4) and the diastolic murmur (DM) of low frequency and low intensity starting just before the third heart sound and continuing through the middle two fourths of diastole

aortic valve, luetic aortitis, or old rheumatic aortic valve lesions The more severe and the more long standing the hypertension, the more likely are the aortic diastolic murmurs to occur (Fig 211)

Apical diastolic murmurs may occur whenever there is marked left ventricular dilatation (Fig 209)

insufficiency may result An accentuated pulmonary second sound develops as pulmonary arterial pressure rises (Fig 210), and, finally, the murmur of tricuspid insufficiency can be heard as the right ventricle fails and the tricuspid valve ring dilates

Aortic diastolic murmurs have been described in arterial hypertension, but they are more likely to be associated with lesions such as a bicuspid

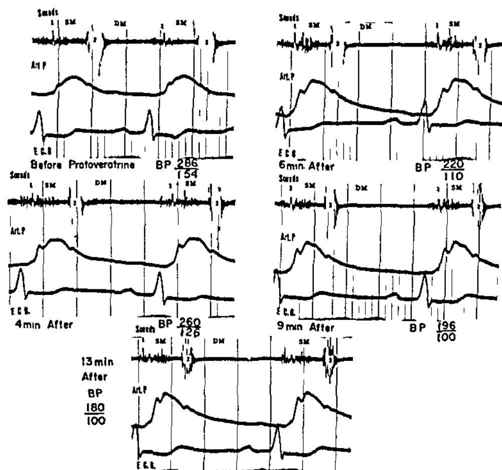


Fig 211 (2 R I S Log) Markedly accentuated aortic second sound and effects of lowering the blood pressure by the administration of protoveratrine Patient had severe arterial hypertension The top left phonocardiogram was taken before protoveratrine was administered Note the low intensity first sound (1) 0 12 sec after the onset of the QRS complex preceded by a group of low intensity vibrations commencing after the peak of the R wave and 0 04 sec after the onset of the QRS This group of low intensity vibrations probably represents the first sound and the single spike labeled 1 is probably an aortic early systolic ejection click It is interesting to note that in the last tracing when the pressure has dropped considerably the low intensity vibrations are more prominent The early systolic click successively diminishes in intensity as the pressure falls throughout the experiment

Note the systolic murmur (SM) of low intensity and medium frequency occupying the first three fourths of systole

There is a very high intensity high frequency second sound (2) which in the first complex lasts 0 12 sec and terminates in a decrescendo manner suggesting some degree of aortic insufficiency In the succeeding tracings after the administration of protoveratrine there is a progressive fall in the blood pressure accompanied by progressive diminution in the intensity and duration of the aortic second sound and the terminal high frequency vibrations

aortic valve, luetic aortitis, or old rheumatic aortic valve lesions The more severe and the more long standing the hypertension, the more likely are the aortic diastolic murmurs to occur (Fig 211)

Apical diastolic murmurs may occur whenever there is marked left ventricular dilatation (Fig 209)

33 / Syphilitic Aortitis and Aortic Insufficiency

Most patients with cardiovascular syphilis are in an age group where other cardiovascular diseases, such as hypertension, atherosclerosis, coronary artery disease, and rheumatic heart disease, are common As a result, syphilitic heart *disease is not diagnosed on the basis of clinical symptoms* but on clinical and laboratory signs The importance of making the diagnosis early and instituting specific therapy before the development of aortic in*sufficiency or formation of an aneurysm is obvious*

SYPHILITIC AORTITIS

In the early stages of syphilitic aortitis involvement of the aorta may be accompanied by normal heart sounds and may not be recognizable clinically, nevertheless, a correct diagnosis is possible in a considerable proportion of cases because of alterations in the heart sounds and the presence of systolic murmurs, secondary to dilatation of the ascending aorta (see figure 94)

AUSCULTATION

The first heart sound usually is normal in timing and in intensity, but it may be muffled in the presence of associated myocarditis An early systolic click secondary to the dilatation of the aorta is common

The second sound may be normal but may be accentuated, with an almost metallic quality, described as tambour On the phonocardiogram this sound is of a higher intensity and a higher frequency than normal and may be of longer duration Systolic murmurs are frequent They are probably formed in the aorta itself rather than at the aortic valve, and usually range from grade 1 to grade 3 in intensity They are loudest early in systole and may be heard clearly both to the left and sometimes to the right of the upper portion of the sternum

SYPHILITIC AORTIC INSUFFICIENCY

*The root of the aorta and the aortic valve ring may be involved as syph*ilitic aortitis progresses, resulting in dilatation of the aortic ring, widening of the commissures, separation of the leaflets and, finally, aortic insufficiency

AUSCULTATION

Depending on the degree of insufficiency, the aortic second sound may remain loud and tambour in quality, it may be diminished, or it may even become inaudible With the onset of failure, the first sound also diminishes in intensity, and both protodiastolic and presystolic gallops may develop The typical decrescendo diastolic murmur of aortic insufficiency can be heard It may be short and quiet in the early stages of the lesion, but later it becomes long and sometimes comparatively loud

The progressive left ventricular dilatation which accompanies aortic insufficiency leads to dilatation of the mitral ring, with the apical systolic and mid diastolic murmurs of mitral insufficiency As both the left and the right ventricles fail, the murmurs of tricuspid insufficiency become apparent

AUSTIN FLINT MURMUR

The Austin Flint murmur (see figure 95) is a presystolic murmur heard at the cardiac apex in patients with aortic insufficiency who do not have mitral valve disease Its authenticity cannot be proved until postmortem examination reveals a normal mitral valve This murmur cannot be confidently diagnosed if the patient has rheumatic heart disease, but it may be strongly suspected in patients who have aortic insufficiency secondary to syphilitic aortitis Many articles have been written suggesting that Flint was describing a mid diastolic murmur, but Flint was very specific in his description of the timing of the murmur

> The murmur occurs just before the ventricular systole or the first sound of the heart it continues up to the occurrence of the first sound and instantly ceases when the first sound is heard[1]

Even though almost a hundred years have passed since the first description of this murmur in 1862, little has been added to the original description of the possible cause of the murmur It is thought that backflow through the aortic valve causes partial closure of the mitral valve, and especially its aortic cusp so that with the onset of atrial systole, a presystolic mitral murmur results This murmur may be accompanied by a thrill

It is impossible to differentiate the Austin Flint murmur from the presystolic murmur of mitral stenosis by auscultation alone

Three factors are essential for diagnosis (1) an apical presystolic murmur (2) aortic regurgitation, and (3) a normal mitral valve

REFERENCE

1 Flint A On cardiac murmurs Am J M Sc 44 29 1862



Index